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THE
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VOL. LIX FEBRUARY, 1949. NO. 2

ESOPHAGEAL LESIONS IN DIFFUSE SCLERODERMA.*†‡

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The frequency with which esophageal lesions appear in generalized scleroderma was drawn to our attention early in 1943. Six consecutive cases were seen at that time in which a disturbance of esophageal function was demonstrated. The symptomatology, Roentgenologic and esophagoscopic findings were reported,[1], and biopsies were made from the lower end of the esophagus in two of the cases.

The occurrence of esophageal lesions in this disease was apparently first reported in 1903, but up until the time of writing our report only 16 cases[2-8] were collected by us in the literature, only one of which had undergone an esophagoscopic examination during life.

Interest was apparently renewed in scleroderma about that time with the realization that lesions of the viscera were common. Two additional reports on involvement of the gastrointestinal tract[9,10] were presented before publication of our report,[1] and shortly thereafter several reports appeared on visceral lesions in scleroderma,[11-17] all of which include reports of esophageal involvement. The variety and importance of

*From the Division of Otolaryngology of the University of Chicago.
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the visceral manifestations of the disease has become generally recognized. The disease holds particular interest for the esophagoscopist because of the clinical and esophagoscopic manifestations and because of certain questions connected with their interpretation.

Since the former report our observations have been augmented greatly by examination of additional patients and by autopsy studies. The gross and microscopic appearance at postmortem examination is of especial value in the interpretation of the clinical, Roentgenologic and esophagoscopic findings.

MATERIAL.

In addition to the six patients included in the 1943 report, 13 cases of diffuse generalized scleroderma* have since been observed. Disturbances of esophageal function have been searched for in 16 of these and the findings briefly listed in Table 1.

TABLE 1.
ESOPHAGEAL LESIONS IN DIFFUSE GENERALIZED SCLERODERMA.

Total number observed since 1942	19
Number with history suggesting pharyngeal or esophageal dysfunction	13
Roentgen ray examination of esophagus made in	17
Positive Roentgen ray evidence of esophageal dysfunction	15
Dysfunction consisting of: Delayed ability of esophagus to cleanse itself of barium; inability to obtain a normal fold pattern and delayed or absent peristalsis	15
Evidence of some degree of stenosis at lower end of esophagus	6
Esophagoscopic examination	11
Apparent stiffening of the esophageal walls plus evidence of inflammatory reaction	11
Leucoplakia, ulceration and some degree of stenosis	5
Gastric type mucosa observed immediately below the stenosed area	4
Ulcerations at lower end of esophagus, proven either by esophagoscopy or at postmortem examination	8

It was pointed out that complaints referable to the esopha-

*The term acrosclerosis has not been used in this or in the former report. The condition which has sometimes been designated as acrosclerosis is considered to be identical with what always has been called diffuse or generalized scleroderma with sclerodactylia.

gus, as well as to the involvement of other viscera, are frequently absent because they are obscured in the early stages by the relatively greater severity of the skin and joint manifestations.

The fact that in nine other cases of diffuse scleroderma seen previous to 1942 no esophageal symptoms were detected, while in the next 19 cases failure to find definite evidence of esophageal lesions occurred in only one of the completely examined cases is due mainly to a more thorough search having been made in the latter group and also to the stage to which the disease had progressed.

The absence of gross esophageal lesions at autopsy in three of the first nine cases observed clinically does not preclude the presence of sclerodermatic changes in the connective tissue and alteration of function of the organ since Roentgenologic and microscopic examinations were not done.

Of the 19 cases seen since the beginning of 1943, eight are known to have died, of which seven were autopsied. Several others, some of which were in advanced stages, could not be traced.

The cause of death in most cases has been the myocardial and the pulmonary lesions.

CASE RECORDS.

In the former report the esophageal symptoms either volunteered or elicited on questioning were described in five cases. The sixth case had symptoms referable to impaired function of pharyngeal musculature. Three of the six cases have since come to autopsy. No follow-up report has been obtained on the other three in the past three years. The 13 cases observed since that report have provided much additional information and the esophageal findings in several selected cases will be described.

Case V. F.: Female with onset of symptoms of scleroderma at about 39 years of age. Skin and joint symptoms appeared first.

Dermatologic Diagnosis: Diffuse generalized scleroderma. Difficulty in swallowing appeared six months after skin symptoms. This consisted of

regurgitation through the nose and coughing on attempting to swallow fluids. Also, food seemed to lodge behind the sternum for some time, and occasionally was brought up some time later. No pain related to swallowing and no heart burn.

Symptoms gradually improved over a period of a year and when examined her symptoms were as follows:

Regurgitation of fluids through the nose on swallowing only occasionally.

Unable to laugh and cough effectively. Difficulty in talking due to loss of air through the glottis. Unable to gargle because the liquid runs down the esophagus.

Unable to swallow either liquid or solid food while lying down.

Examination of pharynx and larynx demonstrated that the glottis did not close adequately on coughing or phonation. Inability to laugh or cough effectively and the difficulty on talking were due to inadequate closure of both true and false cords and resultant air loss. Movements of the larynx and pharynx were symmetrical.

Injection of prostigmine methyl sulfate 1 mg. intramuscularly did not produce any change in the glottic function, but produced cramps and loss of control of bowels and bladder.

Roentgenologic examination by Dr. R. H. Morgan demonstrated "spectacular dysfunction of the pharyngeal and esophageal muscles involving both voluntary and involuntary muscles. Patient experienced extreme difficulty in causing the constrictor muscles of the pharynx to act, and as a result barium flowed up into the nasopharynx and into the larynx when swallowing in the prone position. Normal esophageal peristalsis was absent. There were no tertiary contractions and no evidence of ulceration in the lower end of the esophagus." The patient was not able to perform a Valsalva test satisfactorily, hence the condition of the cricopharyngeus muscle and the presence of a phrenic ampulla could not be determined.

Esophagoscopic Examination: The cricopharyngeal sphincter appeared to function adequately.

There was some difficulty in extending the head due to sclerodermatic changes.

The esophageal lining appeared more pink than normal in the middle and lower thirds, but no leucoplakia, exudate or ulceration was evident.

Comment: Pharyngeal and esophageal dysfunction occurred early in the course of the diffuse scleroderma, the former constituting a major problem.

That the symptoms were due to peripheral disease involving muscular function seems confirmed by the absence of central neurologic signs and also the demonstration of sclerodermatic changes in the esophageal walls on microscopic examination in cases examined since that time.

Difficulty in swallowing which could be related to pharyngeal dysfunction has occurred in three cases of the group.

Case M. S.: Female. Onset of symptoms of scleroderma at about age 65. Skin symptoms involving the fingers were followed in three months by difficulty in swallowing. Food seemed to stick at times and fail to go all the way down. If she attempted to ignore the symptom and continued eating, regurgitation of undigested food sometimes occurred. Liquids went down without trouble.

Dermatologic Diagnosis: Diffuse generalized scleroderma.

Roentgenologic examination by Dr. L. Donaldson revealed the following:

The esophagus was unable to empty itself of barium even with the patient in the vertical position, although the outlet was adequate.

Active peristalsis was practically absent.

A persistent narrowing was present a few centimeters above the diaphragm, with a lumen reduced to about 1.5 cm., with some dilatation above. A good fold pattern could not be obtained in the esophagus above the narrowed region, not because of inadequate outlet but apparently because of mixture of fluid and air.

A suggestion of a small diverticulum was seen in mid-portion.

Evidence was obtained on repeated examination that the region between the constriction and the diaphragm represented stomach extending up through the hiatus for a few centimeters. The coarse fold pattern characteristic of stomach mucosa was demonstrated in this region, particularly with the patient in the prone-oblique Trendelenburg position.

The impression was of a "short esophagus."

Esophagoscopic Examination (Dr. H. B. Perlman): Intravenous pentothal anesthesia. Little resistance at the cricopharyngeus. Mucosa appeared smooth. No ulcers or induration were recognized, but a biopsy of tissue removed from a whitish area near the lower end of the esophagus proved it to be taken from the edge of an ulcer.

Microscopic Examination: The surface epithelium of the ulcerated area was replaced by granulation tissue which showed numerous degenerated polymorphonuclear cells on the free surface and infiltration with these and many small round cells. The granulation tissue contained many small blood vessels and smooth muscle tissue beneath. The remainder of the subepithelial connective tissue was infiltrated with plasma cells and Russell bodies.

Comment: The esophageal dysfunction was an early manifestation of the diffuse sclerodermatic process in this case.

The chief symptom was dysphagia, but the combination of Roentgenologic and esophagoscopic findings demonstrated inability of the esophagus to empty itself even in the upright position, greatly reduced peristalsis, ulceration and beginning stenosis a few centimeters above the diaphragm, and evidence (Roentgenologic) that the stomach extended above the diaphragm. The latter occurrence has been usually interpreted as congenitally short esophagus; however, when the incidence of this finding in generalized scleroderma with esophageal

stenosis is considered, the interpretation of a shortening due to disease appears to be the more probable explanation.

Case R. S.: Female with onset of symptoms of scleroderma at about 50 years of age. Skin and joint symptoms were predominant from the onset. Raynaud-like symptoms present.

Dermatologic Diagnosis: Generalized scleroderma.

For about six years patient had experienced pain in her chest from the epigastrium to the neck, coming on mainly after lying down, sometimes also when in the upright position. Pain had been relieved partly by various antacid preparations. She also had some difficulty in swallowing in that it frequently made her cough. Food went down without any feeling of sticking. The sensation of pain and heartburn came on afterwards.

Roentgenologic examination by Dr. L. Donaldson demonstrated that barium tended to remain in the esophagus. The peristaltic wave was present, although weaker than normal. The fold pattern was obtained with difficulty.

No evidence of ulceration or constriction was observed.

Esophagoscopic Findings: Intravenous pentothal anesthesia. The upper third of the esophagus did not appear abnormal. In the middle third the color was more pink than normal, and in the lower third, near the lower end, patches of leucoplakia were evident with narrow furrows between, in which red granulation tissue formed the base. There was no definite constriction seen. Below this area the mucosa changed abruptly to the gastric type without inflammatory signs. The relation of the upper border of gastric mucosa to the level of diaphragm was not definitely determined.

Comment: The complaint of pain behind the sternum, worse after lying down, has been prominent in at least five of the eight patients known to have ulcerations in the lower third of the esophagus. Although the Roentgenologic examination in this case demonstrated rather an early grade of dysfunction, the epithelial lining of the lower third presented evidence of a relatively advanced chronic esophagitis with leucoplakia and superficial ulcerations. Shortening of the esophagus was suspected on esophagoscopy but was not proven.

Case W. S.: Female with onset of symptoms of scleroderma at about age 62. Raynaud-like symptoms predominated for about five years. About two months before admission, pain in the back between the shoulder blades and shortness of breath began to appear after eating solid foods, which was relieved by regurgitation. Symptoms increased and two weeks before admission she had barium studies, esophagoscopy and biopsy of tissue removed from the lower end. Low grade malignancy was reported to be the impression. Her neck swelled after the esophagoscopy but subsided spontaneously.

Dermatologic Diagnosis: Generalized scleroderma with Raynaud-like symptoms.

Roentgenologic examination of esophagus reported by Dr. Paul Hodges showed a narrowing of the lower end of the esophagus, involving nearly

all of the lower third. This was a fusiform narrowing with smooth walls, and a dilatation of the middle third above it, suggesting stricture rather than neoplasm.

Patient coughed on beginning to swallow barium and was seen to have a large unusually shaped diverticulum coming from the posterior wall of the hypopharynx, extending downward as a long, rather narrow structure

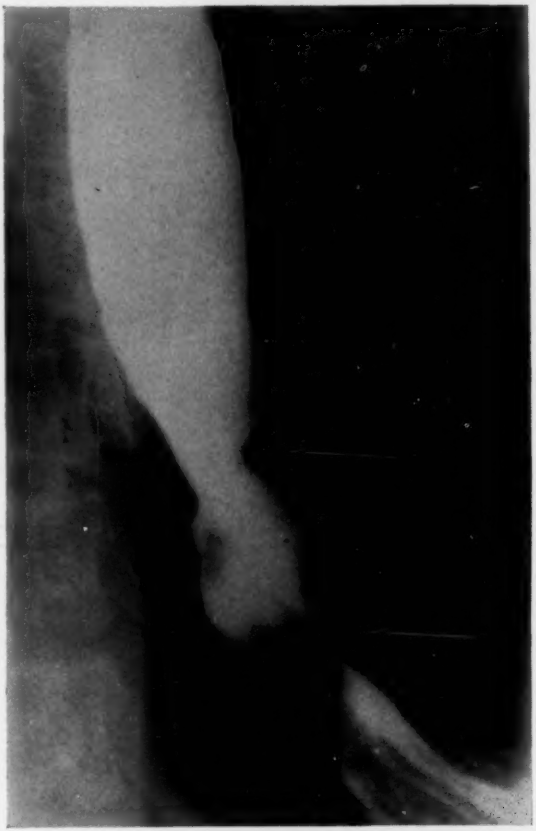

Fig. 1. (Case M.S.) A persistent narrowing occurred at the lower end of the esophagus (upper arrow), with dilatation above. The margins show a fine irregularity in the dilated portion. Barium is not removed. The portion between the constriction and diaphragm (lower arrow) could be made to fill out with patient in the prone oblique Trendelenburg position and demonstrated a coarse fold pattern continuous with that of the stomach mucosa.

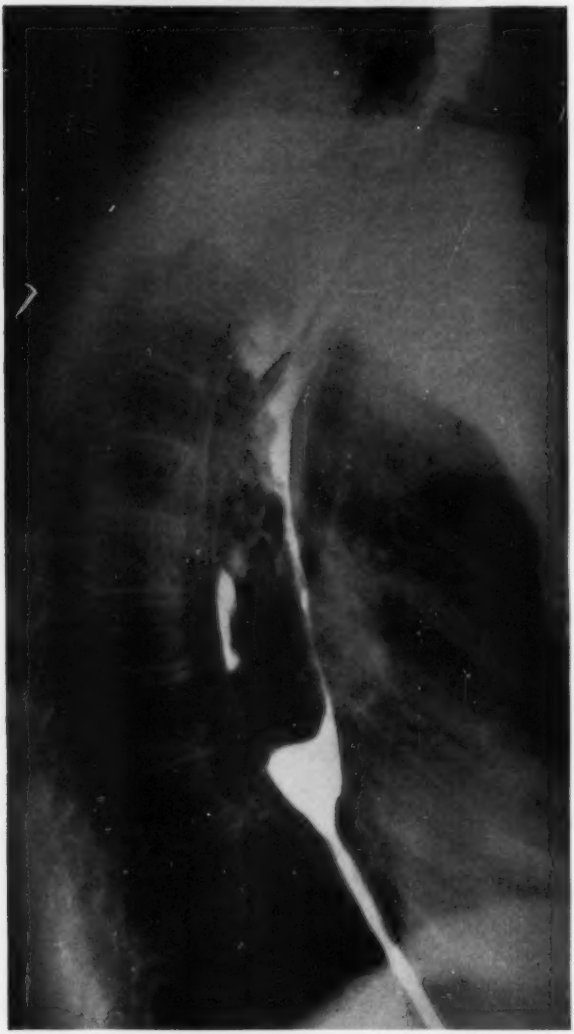

Fig. 2. (Case W. S.) A fusiform narrowing is shown involving most of the lower third of the esophagus with dilatation above. The elongated diverticulum-like structure behind the upper third, containing barium and air, could no longer be seen on X-ray three weeks later. Autopsy examination demonstrated a necrotic tract in this region connected with the lumen of the hypopharynx above and the esophagus below.

containing barium and air to the junction of upper and middle thirds. A possible second small connection with the esophagus was evident near the lower end of the diverticulum (see Fig. 2).

Esophagoscopic examination was not made in view of the former biopsy.

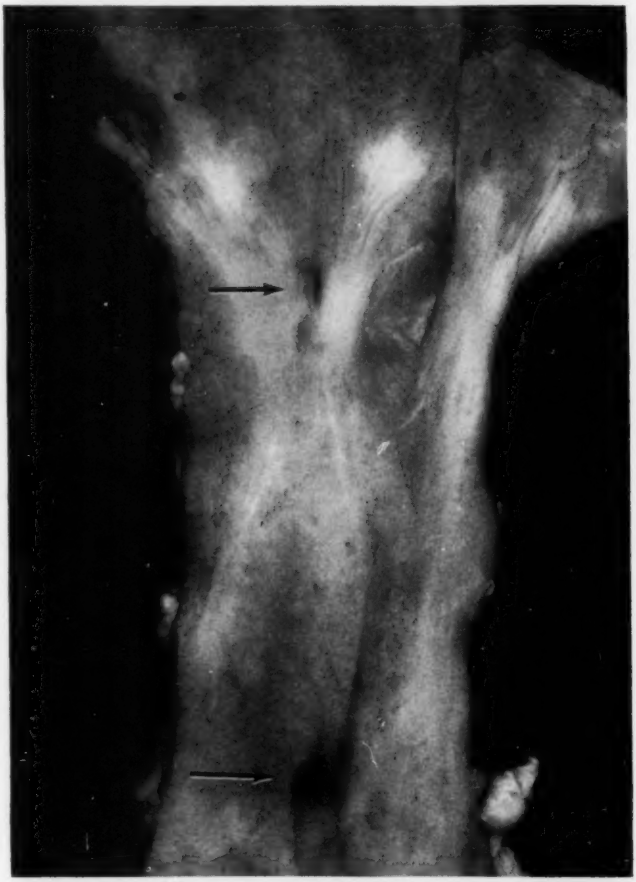

Fig. 3A. (Case W. S.) Shows the two openings in the esophageal wall (arrows). The upper just above the pharyngoesophageal junction, which were connected by a necrotic tract with fibrosed walls.

Fig. 3B. Shows the middle and lower thirds of the esophagus down to the site of the anastomosis. Large hyperkeratotic plaques are evident, the intervening furrows frequently showing superficial ulcerations.

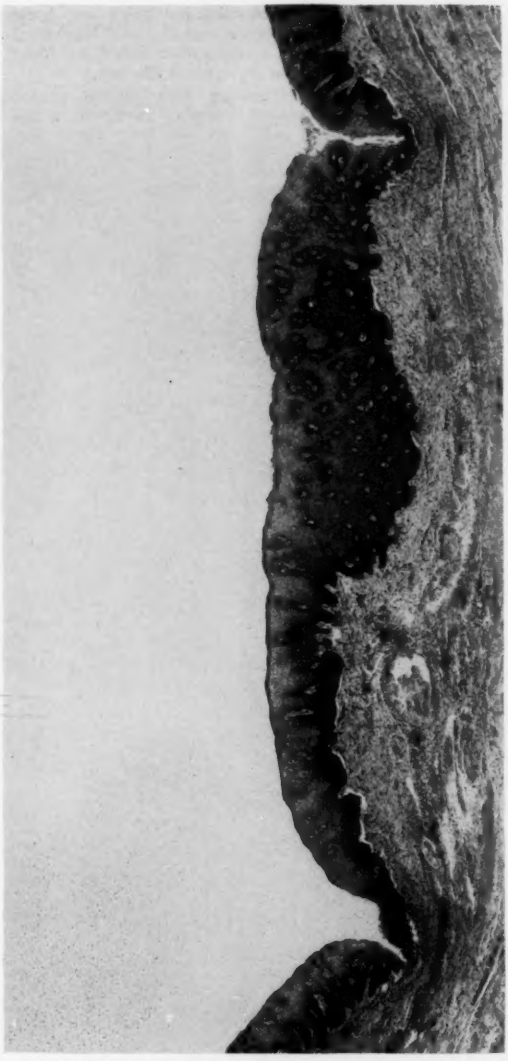

Fig. 4A. (Case W. S.) ×24. Section through hyperkeratotic plaques (leucoplakia) and intervening furrows.

One week later, X-ray examination did not show any barium rests in the mediastinum. At fluoroscopy, three and one-half weeks after the first examination, no evidence of the diverticulum could be found, although thick and thin barium and various postures were used.

Transthoracic esophageal gastrostomy with lateral anastomosis was then carried out. The surgeon noted that the lower end of the esophagus "was slightly narrow and firm, beginning one inch above the diaphragm

Fig. 4B. (Case W. S.) ×26. Upper margin of the diffusely ulcerated and stenosed area in lower third.

and extending upward two inches. No sac-like dilatation was noticeable about it."

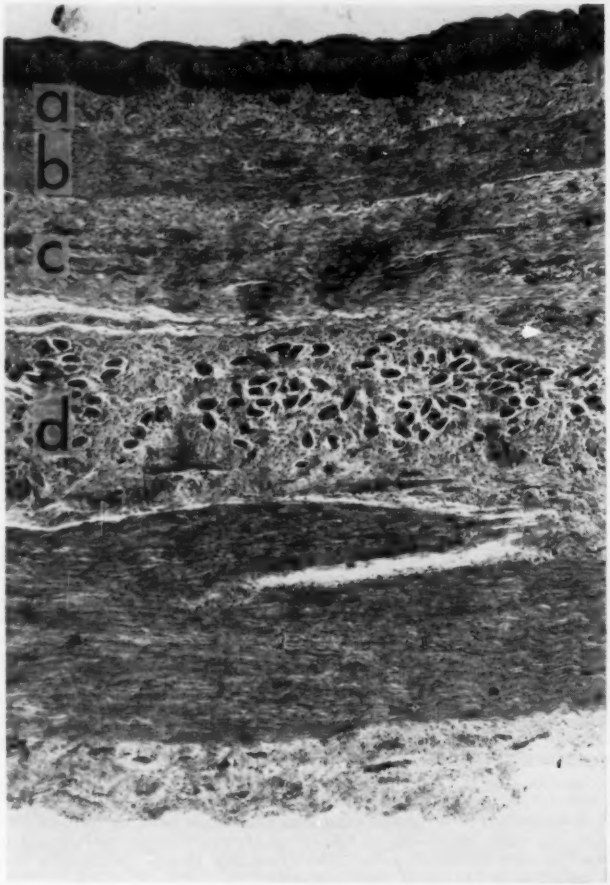

Fig. 5. (Case W. S.) ×70. Section through the upper part of the esophagus. The fibrous connective tissue layers (A) beneath the epithelium and (B) between the muscularis mucosa (C) and inner musculature coat (D) show thickening and homogenization of the fibres. The inner muscular coat contains some striated muscle as well as smooth muscle at this level. Thickening of intervening connective tissue fibres has occurred in this layer, widely separating the degenerated muscle fibres.

The postoperative course was unfavorable. Signs of pneumonia developed, probably due to aspiration. Death occurred on fourth day.

The postmortem examination showed a rather extensive pneumonia. A localized empyema was present at the site of the anastomosis, suggesting that the diseased state of the esophageal wall may have contributed to infection.

The complete case history and autopsy report will be published elsewhere in detail. The gross and microscopic examination of the esophagus is of particular interest to the endoscopist and is briefly reviewed here.

Esophagus — Gross Appearance: There was a small pit in the posterior wall at about, or just above, the junction with the pharynx, and a second pit near the junction of upper and middle thirds, into which a probe could be easily passed (see Fig. 3A).

The middle third presented a diffuse moderate dilatation. The epithelium was diffusely hyperkeratotic, presenting a patchy appearance. The stiffened white patches varied in size up to two-thirds of a centimeter and were separated by narrow furrows, the base of which presented bright red granulation tissue (see Fig. 3B).

Microscopic Appearance: The epithelium in the middle third showed the patches of hyperkeratosis, or leucoplakia, between which the epithelium was lost in narrow furrows, with shallow granulation tissue at the base (see Fig. 4A).

At the lower end the ulceration was more widespread. A thin layer of exudate was present, beneath which was granulation tissue containing some thrombosed vessels, hyperplastic capillaries, polymorphonuclear cells, and many small round cells and plasma cells in the depths. The ulcer extended down into the muscularis mucosa. Extensive fibrosis and scarring were present (see Fig. 4B).

Immediately below the more or less diffusely ulcerated and scarred area was gastric mucosa in which no inflammatory signs were evident (see Fig. 4C).

The subepithelial layers of the esophageal wall presented changes of a striking character. This was most evident in the fibrous connective tissue layers beneath the epithelium and beneath the muscularis mucosa. Thickening and homogenization of fibrous connective tissue fibres corresponding to that which characterizes the skin in this disease was present. Nerve bundles and ganglia were surrounded by these fibres, suggesting the possibility of constriction (see Fig. 5).

The inner circular muscle coat, where the muscle fibres are mostly cut transversely, showed thickening of the intervening connective tissue fibres to the extent that muscle fibres showed all degrees of constriction.

The muscularis mucosa showed similar changes to a lesser degree and the outer muscle layer was least affected.

The sections of the esophageal wall in the upper part demonstrated a tract extending from the pit in the lower hypopharynx to the second pit about three inches lower down (see Fig. 3A). This tract contained no epithelium, the walls consisting of a layer of necrotic tissue surrounded by a granulating and fibrosing area. The tract lay in the connective tissue layer, external to the outer muscular coat.

Comment: This case presented several important features.

The history, Roentgenologic and autopsy findings indicated

a perforation just above the pharyngoesophageal junction at the esophagoscopy two weeks before admission.

Spontaneous recovery occurred, aided probably by spontaneous rupture into the esophagus lower down.

History of a similar occurrence following esophagoscopy was obtained from one case (S. S.) described in our former report. Experience has shown that many cases of generalized scleroderma have stiffness of the neck to such an extent that introduction of the esophagoscope under local anesthesia is made extremely difficult and painful. By the use of general anesthesia the procedure can be made less trying and more safe.

The interference with esophageal function consisting of inability of the organ to clear itself, the loss of normal fold pattern and loss of peristalsis is explained by the effect of the sclerodermatic changes within the walls. These changes result in a general stiffening or loss of pliability, interference with muscular action, and possibly involvement of nerves.

The impressions repeatedly obtained at esophagoscopy in other cases with similar Roentgenographic findings consisting of a loss of pliability of the walls, a leathery consistency, a color more pink than normal in upper parts and patches of exudate, ulceration and stenosis in lower part were explained by the autopsy examination in this case.

The spectacular picture of leucoplakia or hyperkeratotic plaques with intervening ulceration could scarcely be appreciated to the same extent on the limited view obtainable through the esophagoscope.

The sections demonstrate the existence of gastric mucosa up to the ulcerated and stenosed lower end of the esophagus. According to the surgeon's note the esophagus may have extended only to one inch above the diaphragm at the time of operation, the stomach wall extending up through the hiatus.

In addition to the above four cases in which the findings

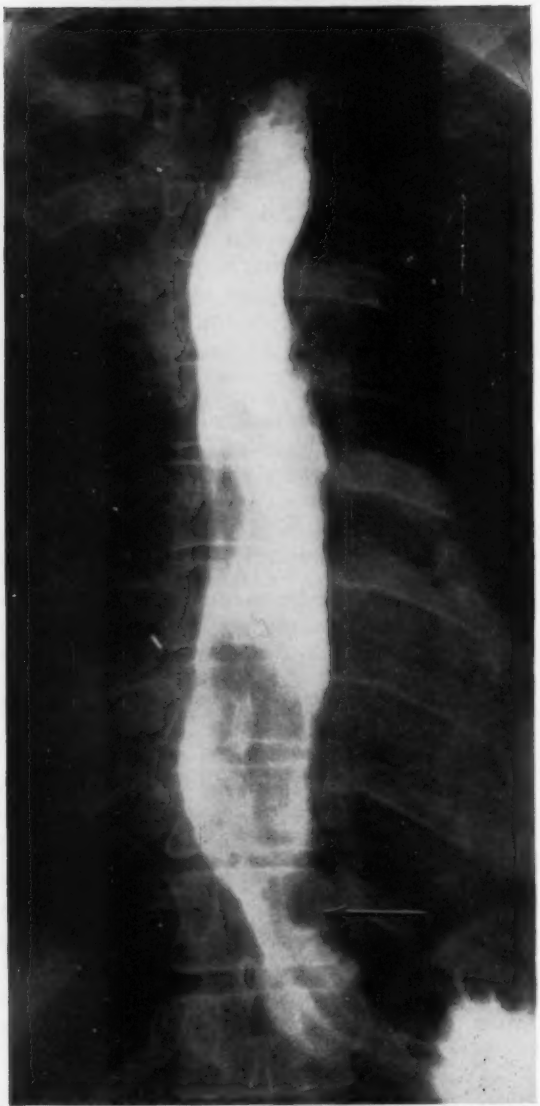

Fig. 6. (Case J. F.) Patient in horizontal position. The esophagus remains dilated throughout. The portion between the stenosis (arrow) and the diaphragm was proven to be stomach wall. (See Jour. A. M. A., 123:745-750, Nov., 1943, Fig. 2 on p. 8.)

warranted a detailed description of the esophageal picture, three of the cases included in the former report have since come to autopsy.

Case N. S. (1) with generalized scleroderma, Raynaud-like symptoms and advanced esophageal involvement was described in the former report. This patient had suffered from burning pain behind the sternum coming on after meals, after lying down and on vomiting and bowel movements for 18 months. Roentgenologic findings showed inability of the esophagus to empty, retention of air mixed with barium, loss of peristalsis and stenosis at the lower end. The stenosis was situated a few centimeters above the diaphragm, at what was thought to be the upper border of the phrenic ampulla (see Fig. 4 of former report). A review of the films in the light of evidence obtained in autopsied cases since that time suggests the interpretation that the stomach extended up through the hiatus and that the esophagus was short. The type of folds seen on X-ray in the portion below the stricture was suggestive of gastric rather than esophageal mucosa.

The esophagoscopic examination, as described formerly, showed gastric type of mucosa immediately below the stenosed area.

Two views (see Figs. 5 and 6 of the former report) showed tissue removed from the ulcerated area near the lower end of the esophagus.

This patient expired on Oct. 2, 1944. The autopsy report obtained from the examining pathologist stated that death had occurred from unresolved pneumonia. The esophagus showed a healed esophageal ulcer at the lower end, an active ulcer just above that and scarification of the mucous membrane for about two inches above the ulcer.

Comment: The esophagoscopic findings one and one-half years earlier corresponded to the postmortem description in that ulceration and scarification were present. The level of the ulcerated area in relation to the diaphragm was not noted in the autopsy description.

A review of the earlier fluoroscopic and esophagoscopic findings strongly suggests that the portion between the stenosis and the diaphragm formerly interpreted as phrenic ampulla consisted of stomach wall extending through the hiatus, and that the esophagus was short.

The phrenic ampulla would, therefore, occupy the area of ulceration and stenosis in the lower third and hence fail to show on examination.

Case F. N. described in the former report was again reviewed. The fluoroscopic interpretation at that time had been that the dilatation which appeared at the lower end of the esophagus represented phrenic ampulla (see Fig. 4, Case 5, of the former report). The esophagoscopic findings were, however, that the mucosa in the lower end of the lower third of

the esophagus showed a thin layer of exudate which when peeled off left a red granulating surface.

Below that area the mucosa corresponded in appearance to gastric mucus and was free from inflammatory signs. The level of the diaphragm in relation to this area was not determined at esophagoscopy.

Review of the Roentgenologic and esophagoscopic findings in this case in the light of information now available indicates that the esophagus was short and that the stomach wall extended up through the hiatus for a few centimeters. The phrenic ampulla of the esophagus would not likely be demonstrable in such a case since it would be in the area most affected by the fibrosis following ulceration.

Case M. G. described in the former report expired on May 11, 1947, after the disease had been present for 17 years. Raynaud's disease had been the original impression, with indications of generalized scleroderma later becoming evident.

At fluoroscopy four years before termination, the slow emptying, loss of primary peristaltic movements and inability to form a normal fold pattern had been noted.

Esophagoscopic examination at that time demonstrated the loss of pliability of the walls and the slightly pink color of the mucosa, but no leucoplakia and no ulceration or stenosis.

Postmortem Examination: Cause of death: pulmonary and cardiac complications of the generalized scleroderma. Esophagus: The mucosa appeared grossly normal. No ulceration or leucoplakia.

Microscopic Examination: The epithelium showed no definite abnormalities. A section through the wall in the lower end showed pathological changes.

The fibrous tissue layers beneath the epithelium and the submucosa showed thickening and homogenization of fibres. The inner coat of circular muscle fibres showed thickening of fibrous connective tissue between muscle fibres and great variation in the size of the latter. The appearance suggested constriction and degeneration of muscle fibres as a result of the fibrous tissue changes.

The muscularis mucosa showed some evidence of the same process, while the outer muscle layer appeared least affected.

Comment: Although impaired esophageal function was demonstrated four years earlier in this case, there were few symptoms and no inflammatory signs involving the surface epithelium. No hyperkeratosis or ulceration occurred.

The lack of pain or burning beneath the sternum and the absence of epithelial changes suggest that the cardiac sphincter may have retained its function in this case. No definite evidence was obtained that shortening of the esophagus was present.

Case J. F. described in the former report was esophagoscoped repeatedly from 1943 to 1947 for relief of the stenosis which was located about 4 cm. above the diaphragm (see Fig. 6). The leathery consistency of the esophageal walls noted in the former report was consistently evident.

Leucoplakia were observed in the lower third with superficial ulcerations in the lower part between the hyperkeratotic patches. More diffuse ulceration was evident at the lower end upon stripping off a layer of exudate with the suction tip.

Immediately below the stenosed area the mucosa was dull dark red in

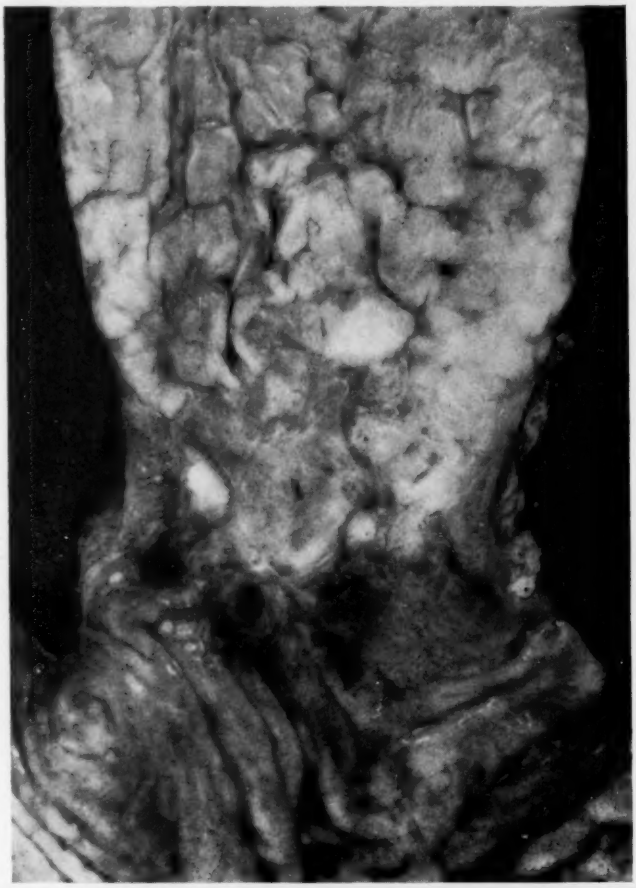

Fig. 7. (Case J. F.) Photograph of the stenosed and ulcerated region at the lower end of the esophagus showing extreme hyperkeratosis above and gastric mucosa below.

color, corresponding to gastric mucosa in appearance, without evidence of inflammatory changes (see former report).

Death occurred on March 3, 1947, from pulmonary and cardiac compli-

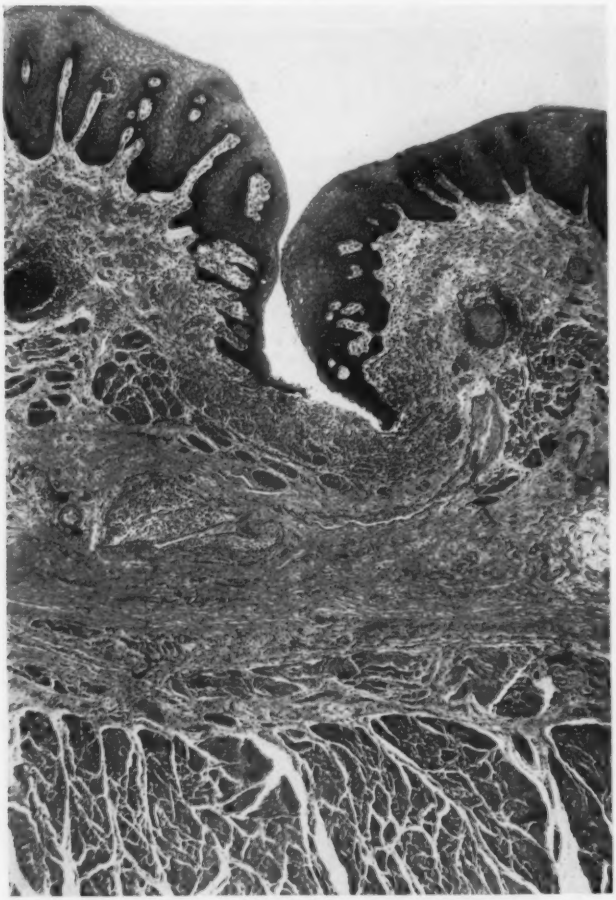

Fig. 8. (Case J. F.) ×60. Lower third of esophagus. Superficial ulceration in the furrow between hyperkeratotic plaques. The thickening and homogenization of fibrous connective tissue beneath the epithelium, beneath the muscularis mucosa, and in the inner muscle layer is demonstrated.

cations of scleroderma. The complete pathologic report will be presented elsewhere.

Postmortem Examination of Esophagus: The esophagus when removed and opened showed extensive hyperkeratosis or leucoplakia in the form

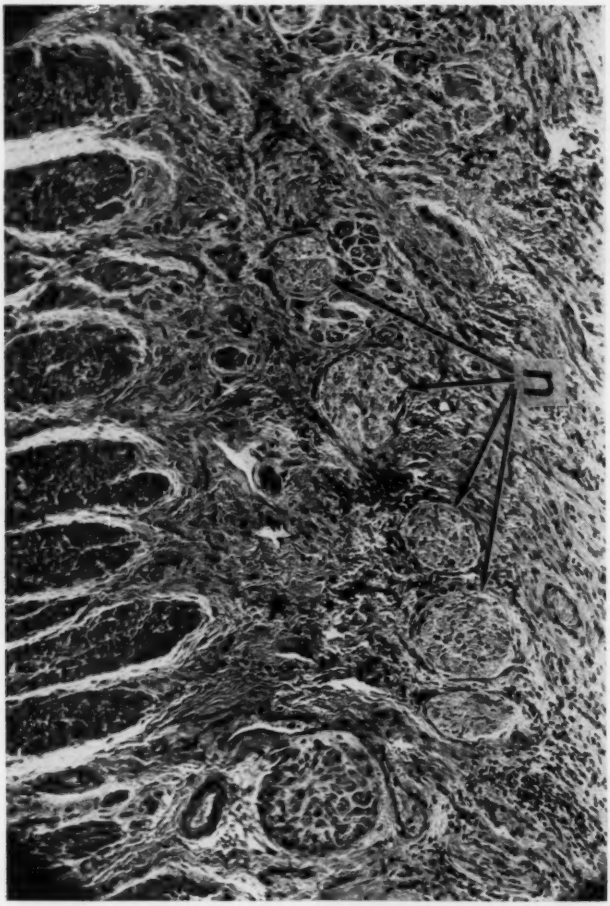

Fig. 9. Case J. F.) ×120. Lower third. A row of muscle bundles below and nerves (arrows) above, surrounded by thickened connective tissue fibres.

of patches, which increased in thickness from the middle third down to the site of the stenosis.

In the furrows between the patches of raised epithelium, narrow bands

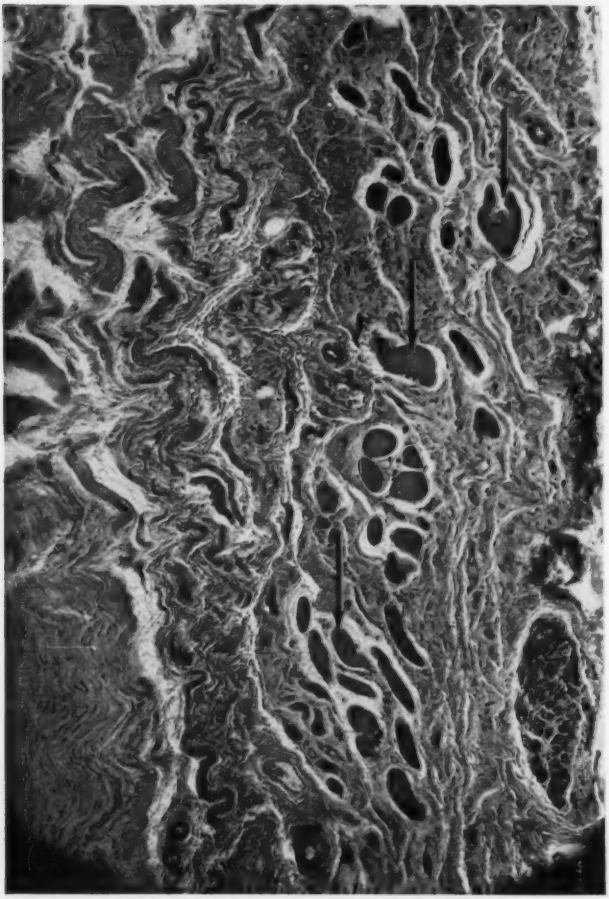

Fig. 10. (Case J. F.) ×120. Upper third. Section through the inner muscle layer and part of the outer coat. The inner coat consists of sparse, smooth muscle cells and both atrophic and swollen degenerating striated muscle fibres (arrows) in a dense collagenic stroma.

of red granulation tissue were evident, with occasional larger ulcerated patches at the site of stenosis, covered in places by a layer of exudate.

Caudal to the stenosed area, the lining consisted of gastric mucosa, thrown into folds without gross evidence of inflammatory reaction. The gross appearance of the esophagus is illustrated in Fig. 7.

Microscopic Examination: The epithelium of the lower third of the esophagus showed irregular patches of leucoplakia, increasing in thickness towards the lower end, with narrow furrows in the base of which superficial ulceration has occurred (see Fig. 8).

Some large ulcers were present at the lower end, with more extensive loss of epithelium and of submucosal connective tissue extending down into the muscularis mucosa in some areas. There was infiltration by round cells, neutrophilic and eosinophilic polymorphonuclear cells, proliferation of fibrous connective tissue and scarring. An occasional thrombosed vein was seen in the ulcerated area. A thin layer of exudate was present on the surface in some areas. Immediately below the stenosed area gastric mucosa was present.

The subepithelial layers of the esophagus showed striking pathologic alterations throughout. These were most evident in the fibrous connective tissue layers beneath the epithelium and beneath the muscularis mucosa. Thickening and homogenization of the connective tissue fibres was present, producing a more dense and broad appearance of these layers (see Fig. 8). The same process was evident surrounding nerves and their ganglia (see Fig. 9). These changes resembled the findings in the skin which were typical of the disease. The layer which showed the next most evident change was the inner muscular coat. In this layer the fibrous tissue between muscle fibres showed the same thickening with varying degrees of degeneration of the muscle fibres. The layer consisted mainly of the altered fibrous tissue at the expense of the muscle fibres which were separated and showed varying degrees of reduction in size (see Fig. 10). The muscularis mucosa showed the next most evident change, corresponding to the inner circular muscle layer, but to a lesser degree. The outer muscle layer showed relatively little alteration.

Comment: Disturbance of function, consisting mainly of loss of primary peristalsis, delayed emptying, particularly when in the lying position, and moderate stenosis had been evident on Roentgenologic examination four years before death.

The exisence of chronic esophagitis, with superficial ulceration near the lower end of the lower third covered by an exudate, scar tissue contraction, and the existence of gastric type of mucosa were recognized on repeated esophagoscopy. The esophageal walls had also exhibited a loss of pliability on passing the instrument.

The extent and distribution of the surface lesions shown on the gross pathologic examination was more striking, however, than could be realized from the esophagoscopic view.

In the previous report the Roentgenologic impression had

been that the stenosis occurred above the phrenic ampulla of the esophagus.

The autopsy findings demonstrated that gastric mucosa had extended up through the diaphragm for several centimeters to the stenosed area. That part of the esophagus which normally forms the phrenic ampulla was, therefore, the site of the ulceration and stenosis and would not be demonstrable on X-ray. The interpretation suggested by the pathologic picture was that the esophagus had been shortened by the pathologic fibrous tissue changes within its walls and the stomach pulled up through the hiatus to that extent.

The extensive involvement of fibrous connective tissue, and secondarily of muscle fibres, provides an explanation for the disturbance of function. Possibly nerve involvement by the same process may also have been a factor (see Fig. 9).

The alterations in the epithelial layer probably represent a combination of trophic changes with superimposed surface irritation and inflammation. As was previously suggested, the sclerodermatic process may through impairment of muscular function lead to regurgitation of gastric contents which are then inadequately removed, causing pain and tending to produce inflammatory changes, or a diffuse esophagitis. The leucoplakia, ulceration and stenosis appear to be best explained as the direct result of a diffuse chronic peptic esophagitis.

GENERAL DISCUSSION.

A complaint referable to the swallowing mechanism or to the esophagus was volunteered only in those cases with fairly severe symptoms. In many cases the severity of the skin and joint symptoms obscured mild difficulties with swallowing.

Of the last 19 cases, six gave no history of swallowing difficulties.

Five gave a history indicating pharyngeal or glottic dysfunction such as difficulty in starting solid food through the pharynx, coughing on swallowing fluids, inability to close the

glottis adequately, or regurgitation through the nose on swallowing liquids.

Six gave a history of pain which could be related to the esophagus.

Eight cases complained of dysphagia described as inability to swallow in the lying position, a feeling as of food lodging behind the sternum, regurgitation of swallowed food after the meal or inability to get solids down. One case already had a gastrostomy when first seen.

The varying degrees of pathological findings in the esophagus on Roentgenological examination were as follows:

In all cases except two there was failure of the esophagus to clear itself of barium in either the lying or the upright position, retention of air bubbles with barium, fine irregularities along the margins, inability to demonstrate a normal fold pattern, and impairment and eventually absence of primary peristaltic waves.

Stenosis was demonstrated to a varying degree in the lower part of lower third with moderate dilatation above in five of the advanced cases and probably present in a sixth. The stenosis occurred always a few centimeters above the diaphragm. In three of these it was possible to demonstrate a fold pattern extending up to the stenosis which is characteristic of gastric mucosa rather than the finer folds characteristic of the phrenic ampulla of the esophagus. Repeated examination was sometimes necessary to bring out the fold pattern.

Of the 19 cases seen since 1942, esophagoscopic examination had been done in 11 cases. In some, repeated examinations were done for the purpose of observation and dilatation of the stenosed area.

All exhibited some degree of abnormality. In four this was limited to an alteration in color of the mucosa in the lower part and a loss of normal pliability throughout. In addition to these characteristics seven cases showed surface changes in the lower third consisting of exudate and ulceration. Leucoplakia was evident in some and stenosis was present in four.

When esophagoscopic and autopsy findings are combined there were 14 cases in which the esophagus was inspected directly. Eight of these presented ulcerations at the lower end. Five of the eight had a definite stenosis, and a sixth a probably early stenosis.

A characteristic of the stenosis in these cases was that it occurred always a few centimeters above the diaphragm. The esophagoscopic examination demonstrated a fairly abrupt change in the mucosa below the stenosed or ulcerated area to the gastric type, and it was possible in three to obtain Roentgenological evidence that the stomach wall extended up to the stenosis. Proof was obtained at autopsy in two that gastric mucosa extended to the lower border of the ulcerated and stenosed area. It, therefore, appears to be a characteristic of those cases of diffuse scleroderma with ulceration and stenosis at the lower end of the esophagus that the esophagus is short and the stomach pulled up through the hiatus.

The impression of "congenitally short esophagus" and extension of gastric wall a few centimeters above the diaphragm has been reported[12,15] in a number of cases on the basis of Roentgenological examination.

Similar cases presented by us formerly[1] were interpreted as having stenosis at the upper border of the phrenic ampulla of the esophagus.

Further observations since that time, including esophagoscopic and autopsy findings, have demonstrated beyond doubt that the area between the stenosis and diaphragm consists of stomach wall. The interpretation that the short esophagus represents a congenital malformation in such cases does not seem tenable. The fact that a shortened esophagus and extension of gastric wall above the diaphragm is so frequent as to be considered a characteristic in generalized scleroderma with ulcerative esophagitis is evidence that the shortening has resulted from fibrous tissue contraction within the walls of the esophagus. While the sclerodermatic process may predispose to shortening, the evidence so far indicates that the fibrosis secondary to ulceration may be the chief cause.

"Congenitally short esopahgus with thoracic stomach" has been frequently reported in the literature without associated diseases such as scleroderma, and in such cases there has usually been some degree of stenosis at the lower end of the esophagus. The explanation has usually been given that ulceration and stenosis result from the irritative action of gastric juice, consequent to the lack of protection afforded by the pinchcock action of the diaphragm.

These findings in scleroderma raise the question as to whether the shortening of the esophagus may not have been secondary to a fibrosing lesion in the esophagus in some such cases, rather than a congenital anomaly.

Great difficulty is sometimes experienced in obtaining sufficient relaxation to permit introduction of the esophagus in some cases of generalized scleroderma, due in part to tightening of the skin of the neck and face and in part to limitation of movement at joints.

Positive evidence was obtained that a perforation of the esophagus had formerly occurred in one case and a history of a probable perforation in another, probably as an indirect result of the difficulty created by the scleroderma. Both healed spontaneously.

Some type of general anesthesia is, therefore, conducive to greater safety in carrying out the procedure in the presence of diffuse generalized scleroderma.

SUMMARY.

The disease known as generalized scleroderma is a disease of fibrous connective tissue in which the skin and joints are usually affected first and give rise to the most distressing symptoms.

Visceral lesions are also characteristic of the disease and involvement of the lungs, heart, kidneys and gastrointestinal tract is evident in varying degree.

Disturbance of the swallowing mechanism, first described

in 1903 and reported in a few cases between 1931 and 1937, has become recognized as a common characteristic.

In 17 cases observed since 1942, pharyngeal or esophageal involvement was demonstrated by Roentgen examination or esophagoscopic examination, or both, in 15. Two additional cases did not have a special examination of the esophagus.

Various stages of pathological disturbance have been demonstrated and the characteristic Roentgenologic and esophagoscopic signs presented.

The observations which were presented in a former report consisting basically of Roentgenologic evidence of loss of function, esophagoscopic appearance and biopsies from ulcerated areas have been augmented by gross and microscopic postmortem examinations.

Fibrous connective tissue changes, similar to those occurring in the skin, have been shown in the walls of the esophagus.

Alteration of function of the esophagus is explainable upon the basis of stiffening, interference with muscular action, and possibly nerve involvement.

The surface changes consisting of hyperkeratosis or leucoplakia, ulceration and stenosis appear to require a factor of surface irritation in addition to probable trophic changes for their production since they are not constant.

This surface irritation is probably supplied by regurgitated gastric juice from which the esophagus is unable to free itself.

The postmortem findings in two cases with stenosis, as well as the accumulated Roentgenologic and esophagoscopic evidence in four additional cases with varying degrees of stenosis, appears to have established the presence of gastric mucosa between the stenosed area and the diaphragm, a distance of 2 to 5 cm.

Circumstances appear to indicate that this represents a

shortening of the esophagus by disease rather than a congenitally short esophagus.

Two factors are present which may contribute to the shortening, the diffuse sclerodermatic process throughout the esophagus and the localized ulceration and fibrosis at the lower end.

Dr. Eleanor M. Humphreys, of the Department of Pathology of the University of Chicago, has aided with the interpretation of pathological material. Dr. Lillian Donaldson, of the Department of Roentgenology of the University of Chicago, has reviewed the Roentgenological interpretations.

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Footnote: Since this paper was completed, five additional cases of diffuse generalized scleroderma have been examined. In four of these, esophageal involvement of varying degrees was demonstrable on X-ray and esophagoscopic examinations.

MISSISSIPPI VALLEY MEDICAL SOCIETY MEETS AT ST. LOUIS, SEPT. 28, 29, 30, 1949.

The Fourteenth Annual Meeting of the Mississippi Valley Medical Society will be held at the Jefferson Hotel, St. Louis, Mo., Sept. 28, 29, 30, 1949, under the presidency of Dr. Alphonse McMahon, of St. Louis University. At the recent current meetings of the Society and board of directors, the following officers were elected: Dr. Nathaniel G. Alcock, Iowa City, Iowa, President-elect; Dr. Wendell G. Scott, St. Louis, Mo., First Vice-President; Dr. Charles F. Harmon, Springfield, Ill.; Second Vice-President; Dr. John I. Marker, Davenport, Iowa, Third Vice-President; Dr. Harold Swanberg, Quincy, Ill., Secretary-Treasurer; Dr. Ralph McReynolds, Quincy, Ill., Accounting Officer.

FURTHER STATISTICAL STUDY OF AUDITORY TESTS IN RELATION TO THE FENESTRATION OPERATION.*

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In January, 1945, a series of tests of hearing for pure tones and for speech was begun on all patients on whom the fenestration operation was performed by one of us (TEW). Tests were performed before and at various intervals after operation on both the operated and the unoperated ears. Two studies of the inter-relationships of the results of these tests and the Social Adequacy Index for Hearing (SAI) on the first 161 cases have already been published.[10,3] The present report deals primarily with the results on the 55 additional cases operated between Oct. 25, 1946, and Nov. 25, 1947, inclusive. The mean age of the patients in this series was 33.4 years at the time of operation. The standard deviation of the mean (σ) is ± 8.1 years, and the range from 17 to 57 years.

I. – PROCEDURE.

On Oct. 8, 1946, the testing routine was modified and abbreviated. The new routine was planned particularly *a.* to compare the Threshold of Intelligibility for Connected Discourse with Test No. 9 and with "PB" articulation scores, *b.* to compare pure-tone hearing loss with loss for speech, and *c.* to

*This statistical study was carried out under Contract N6onr-272 between the Office of Naval Research and Central Institute for the Deaf.

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evaluate the results of fenestration by means of the Social Adequacy Index. The sequence of tests comprised

1. Pure-tone audiometry by air and bone conduction, performed at McMillan Hospital (Washington University) according to the usual clinical routine;

2. The following monaural, air-conduction speech tests at Central Institute for the Deaf:

   a. Threshold for Speech by Test No. 9 (spondaic words).[6]

   b. Threshold of Intelligibility for Connected Discourse (TICD).[5] In this test the subject adjusted the intensity of a recorded newscast until he could just easily follow the sense of the material.

   c. Articulation score for phonetically balanced monosyllables (PB word lists[4] as recorded at Central Institute for the Deaf), delivered at high intensity. The usual level was 110 db re 0.0002 dyne per cm^2, although for some seriously hard-of-hearing ears 120 db was also used and for others, following fenestration, 100 db sufficed. If more than one level was used the higher score was taken as the "PB max." The object of the test was to measure the *maximum* articulation score (PB max.) that the subject could attain.

   d. Articulation score (PB lists) at the level of the patient's Threshold of Intelligibility for Connected Discourse.

   e. From the hearing loss for speech measured by No. 9 or TICD or the average of the two, and from the "discrimination loss" (100 – PB max.) the Social Adequacy Index was obtained by the tabular method described elsewhere.[3]

3. Free Field Tests, binaural, served chiefly for practice before the monaural tests.

The usual order of the tests was

1. Free Field: No. 9, one test; TICD, 3 settings.
2. Right Ear: No. 9, 1 test; TICD, 3 settings.
3. Left Ear: No. 9, 1 test; TICD, 3 settings.
4. PB list at high intensity followed by a PB list at the Threshold of Intelligibility for Connected Discourse as determined in the previous tests. The same sequence was followed: Free Field, Right Ear, Left Ear.

The entire testing session lasted about one and one-half hours.

The data so obtained allow us to calculate 1) several aspects of the reliability of the various tests as applied to hard-of-hearing ears of young and middle-aged adults, 2) inter-relationships between the several tests, 3) the effects of the fenestration operation (as measured a few weeks to several months following operation) and the significance of changes in operative technique and of the type of bone encountered, 4) several other minor comparisons, such as the relation of the gain from fenestration to clinical diagnosis.

II. – RELIABILITY.

1. *Correlation coefficients and standard errors of measurement.*

Descriptions of these statistical measures, with references, have been given in our previous report,[10] and need not be repeated in detail. It will be recalled, however, that the standard error of measurement gives the range above and below the true score within which for the average subject a single test may be expected to fall two-thirds of the time on repeated tests.

Test-retest reliability was calculated for the *unoperated* ear as the Pearson product-moment correlation coefficient *(r)* between the pre-operative and the first post-operative test. The coefficients *(r)* obtained in this way from our data are expressions for the stability of these test measures over a

period of time. They should thus be distinguished from other expressions for reliability which have been used.[1] No allowance is made here for learning effects. A value of r above .80 indicates good reliability and a value above .60 is fair.

The values of r for the various tests are shown in Table I. Some of them, notably for No. 9 and the Social Adequacy

TABLE I.
TEST-RETEST RELIABILITY AND
STANDARD ERROR OF MEASUREMENT OF AUDITORY TESTS.

	Present Series			Previous Series		
	N°	r	σ_m†	N	r	σ_m
Unoperated ear						
No. 9	50	.87	3.1 db	146	.89*	3.9 db
TICD	50	.83	3.6			
PB Maximum*	55	.61	6.2 %	100	.68	10.5 %
SAI	55	.87	4.8 %	156	.83	6.7 %
Air audiogram (average 512, 1024, 2048, 2896)	55	.59	5.9 db			
Gains from Fenestration‡						
No. 9	50	.79	4.3 db			
TICD	50	.71	5.2 db			
Average of No. 9 and TICD	50	.86	3.4 db			

° N = number of cases.
* PB score at 110 or 120 db in present series, at 100 db in first series.
† Standard error of measurement.
‡ Estimated as explained in text.

Index, are very satisfactory and agree well with our previous results. For "PB maximum" the reliability is fair, but here a single figure does not tell the whole story. As we shall see below, the PB maximum scores are very stable if they are high, *i.e.*, 85 or better, but vary much more if the original score is below 85.

The standard error of measurement of the hearing loss for the pure tones most important for speech, even for the average

of four tones, is nearly double the standard error of measurement for Test No. 9. The test-retest reliability is only fair.

For the test-retest reliability of the gains following fenestration it is obviously impossible to get a true test-retest measure. The estimates shown in Table I were obtained by assuming that the reliability of the test in the operated ear was similar to that in the unoperated ear. Since the reliability is expressed as a correlation coefficient, corrections were made where necessary for a difference in the range of the test scores in the operated ear.[8] With these data and the standard deviations and the correlation between pre-operative and post-operative tests on the operated ear we estimated the reliability of the gains by the method of Jackson and Ferguson.[7] The reliability of the average of No. 9 and TICD gains was estimated by using the formula for the reliability of the sum of two scores.[7]

2. Drift or Learning.

To detect the presence of drift, such as might occur from learning or practice effects, the means of the scores on three

TABLE II.
DRIFT.

Test	N	Drift	Significance
Unoperated ear			
No. 9	50	+0.4 db	Not significant at 5% level ("t" test)
TICD	50	+1.0 db	Not significant at 5% level ("t" test)
PB Score at 110* db			
All cases	55	+4.4%	*Significant* at the 1% level ("t" and sign tests)
Scores 85 and over	30	+1.3%	Not significant (sign test)
Scores below 85	25	+8.2%	*Significant* beyond 1% level (sign test)
Operated ear			
PB Score at 110* db	55	+1.7%	Not significant at 5% level ("t" test)

*See Procedure, 2c.

of the tests on the unoperated ears were calculated for the first and for the second sessions. The differences are given in Table II. A positive value indicates that the subjects heard better on the second test than on the first. It is clear that for Test No. 9 and for TICD there is no significant drift. Our previous evidence[10] concerning drift for Test No. 9 was equivocal.

For the unoperated ear the PB articulation score at high intensity (presumably the maximum score that the subject could attain) shows an improvement of 4 per cent on the average. The drift is significant in the sense that the probability of a drift of this magnitude occurring by the operation of chance alone is less than one in a hundred. The significance of the drift is confirmed by the "sign" test also.[10] An improvement of this sort as a result of learning over the first few trials is reasonably to be expected with the PB word lists. The direction of the drift is opposite to the drift that we found in the first series.[10] We have no explanation for the earlier result, and have more confidence in the present series because the word lists were administered at an average of 110 db instead of 100 db and therefore presumably measure more truly the subject's maximum PB score. Furthermore, the standard error of measurement (σ_m in Table I) for PB score at 110 db is 6.2 per cent in the present series as against 10.5 per cent in the first series.

Further light on this situation is shed by breaking down the subjects into two groups, those with high (85 or higher) PB scores at 110 db and those with scores below 85. The group with initially high scores show quite stable PB scores, with an average upward drift of only 1.5 per cent. The group with lower initial scores is much more variable and the average upward drift is 8.2 per cent.

The "t" test shows that the drift of the PB scores for the unoperated ears as a group is significant at the 1 per cent level of confidence, and this result is confirmed by the "sign" test. The same tests show, however, that the slight improvement for the operated ears is not significant at the 5 per cent

level of confidence. Possibly the operation reduced the discrimination of the operated ears slightly, just enough to offset the learning effect.

We conclude tentatively from all of the above information that 1) initially high PB scores at 110 db do not drift significantly and would show a reasonably small standard error of measurement, and 2) initially low PB scores at 110 db would show a considerably larger standard error of measurement. The opposite drifts in the two series seem to offset one another although each in itself is statistically significant. The levels used in the two series were not identical, however. We should expect a slight learning effect, but more data are needed for a final conclusion.

III. – INTER-RELATIONS OF TESTS.

1. Speech Tests and Pure-Tone Audiometry.

The correlation was computed between the hearing losses for speech by Test No. 9 and the average hearing loss by air for the tones 512, 1024, 2048 and 2896 cps. The pre-operative tests for both the unoperated and the operated ears were used, 110 ears in all. The coefficient r is $+.74$. This is exactly the same value that we obtained in the first series.

When this coefficient is corrected for the unreliability of the two tests the estimate of correlation becomes 1.00.[8, p. 203] The two tests apparently measure essentially the same thing although No. 9 is a more reliable measure.

2. Test No. 9 and TICD.

The correlation between the results of Test No. 9 and the Threshold of Intelligibility for Connected Discourse is $r = +.86$ ($N = 110$). This is very nearly as high as the test-retest reliability of Test No. 9 itself, and when corrected for the unreliability of each test the estimated correlation becomes 1.00. TICD may therefore be added to Test No. 9, Test No. 12, and Test 4C as the fourth member of a group of tests of hearing loss for speech that are practically interchangeable with one another. The choice between them depends on the

reliability of each, and even more on their suitability for a particular patient for psychological reasons or because of his educational background.

The mean difference in absolute threshold (not hearing loss) between Test No. 9 and TICD is 2.4 db for 81 values from unoperated ears and 3.3 db for 81 values from operated ears. In each case the TICD threshold is higher than the threshold for No. 9. The difference of about three decibels is slightly greater than the one decibel reported by Falconer and Davis for normal and for hard-of-hearing ears and is probably a better indication of the difference to be expected when TICD is used in a clinical routine.

We had hoped to improve the accuracy of our measurement of hearing loss for speech by adding TICD to the battery. It now appears, however, because of the higher reliability of Test No. 9, that the advantage is slight. If greater accuracy and assurance are required they are better obtained by giving Test No. 9 twice rather than by giving No. 9 once and TICD once. We therefore in October, 1947, eliminated TICD from our routine for fenestration patients and now rely entirely on Test No. 9.

3. TICD and PB Score.

The exact relationship between a test of the threshold for speech and the PB articulation score is of considerable significance as one of us has fully explained elsewhere.[3,2] If we know the PB score that a subject may be expected to get when given a PB list at the level which we have measured as his threshold by another test then *we may use that other test to locate the position of the foot of his articulation curve more accurately or more rapidly than by giving actual PB lists.*

In the present series the PB articulation score at the Threshold for Intelligibility for Connected Discourse was determined directly on operated and unoperated ears. The mean of 81 values of equivalence for operated ears was 25.6 per cent, for 81 unoperated ears was 24.9 per cent. In other words, at the level at which these patients understood only one quarter

of the PB (monosyllabic) words they understood, satisfactorily to themselves, an actual radio newscast. This is an important and convenient equivalence to bear in mind.

The slope of the lower half of the PB articulation curve, using our apparatus and recordings, is 3.85 per cent per db. Applying this relation to the difference in threshold (for the present group of subjects) between TICD and Test No. 9 we conclude that for them the PB score at the No. 9 threshold is 15.3 per cent correct. This value, determined for hard-of-hearing ears, is a little but only a little higher than the value of 8 per cent that we have given as our best overall estimate for normal and for hard-of-hearing listeners combined.[3] On the decibel scale the discrepancy amounts to about two decibels.

4. PB Maximum Score and Clinical Grouping.

The cases were grouped by one of us (TEW) on the basis of clinical tests and pure-tone air and bone audiometry, but in ignorance of the results of the speech tests, into three groups: conductive (C), conductive-mixed (CM), and mixed-nerve (MN). The basis of the classification is the amount of nerve involvement or perceptive deafness believed to be present. The series did not include any cases of pure nerve deafness.

TABLE III.

PB MAXIMUM SCORE AND CLINICAL GROUPING.

	N	Mean PB score at 110 db
Conductive	34	84.9%
Conductive-mixed	18	78.4%
Mixed-nerve	3	65.3%

Tri-serial eta = 0.54, significant beyond the 1 per cent level.

The numbers of cases and the mean PB scores at 110 db for each group are shown in Table III. There is a clear relationship to the clinical grouping, and the "tri-serial eta"[10] calcu-

lated from these data shows that the relationship could not be expected to occur by chance alone once in a hundred times. The tri-serial eta is 0.54 as compared with 0.43 in the previous series.

The result is consistent with our generalization that severe high-tone hearing loss, particularly of the perceptive type, causes a discrimination loss, *i.e.*, a reduction in the patient's maximum PB score.

IV. – EFFECTS OF FENESTRATION.

1. Gains for Speech and Pure Tones.

In Table IV are given the mean gains for speech and for pure tones that were produced by the fenestration operation.

TABLE IV.
GAINS IN HEARING FOLLOWING FENESTRATION.

Test	N	Mean Gain	Confidence Limit
Speech Tests			
No. 9	50	24.3 db	±2.7 db
TICD	50	26.7 db	±2.8 db
Average No. 9 and TICD	50	25.5 db	±2.6 db
Air Audiogram			
512	55	23.2 db	
1024	55	24.7 db	
2048	55	21.5 db	
2896	55	9.5 db	
Average	55	19.7 db	
Average of the 50 cases tested by No. 9 and TICD	50	19.9 db	±3.2 db

The gains are based on the first post-operative test, usually between three and four months after operation. (Our data are still too scanty to make a detailed analysis of the permanence of these gains over longer periods, although we can state broadly that the gains are being retained much better,

although not completely, now that the cartilage stopple is no longer employed.[9])

The figure in the column to the right of the mean gain gives the confidence limits. The confidence limits[8, p. 137] are the limits within which we can expect the true *mean* gain to lie 95 per cent of the time, assuming of course that the selection of cases and operative procedures remain the same.

The gains measured by No. 9, by TICD, and for the first three pure tones are substantial (about 25 db) and quite consistent. The "sign" test shows that the differences between the average of No. 9 and TICD are not significantly different (even at the 25 per cent level) from the gains for any of the first three pure tones. Neither are the differences in gain among these first three tones significant. *The only significant differences are those which involve the frequency 2896,* either alone or in the average audiogram. Here the gain is significantly less (at the 1 per cent level) than the gain by the average of No. 9 and TICD. The gain for 2896 is also significantly less than for the frequency 2048. *The relatively small gain at 2896 is obviously responsible for the smaller gain measured by the average air audiogram which we reported for the previous series*[10] *and confirm in Table IV.*

We must point out that in a majority of our present cases, due to oversight by our testers, the loss at 2896 was not measured directly in one or both of the tests. In these cases the loss at 2896 was interpolated in the audiograms on the basis of the losses measured at 2048 and 4096 and following also the contour of the audiogram for that patient in the tests in which the loss at 2896 was actually measured. From inspection of the audiograms it is clear that the gain at 4096 also tends to be less than for the lower frequencies. However, the average gains for 2048, 2896, No. 9 and TICD have been calculated separately for the 11 cases in which 2896 was directly measured both before and after operation. In spite of the small number of cases the "t" test shows that the differences stated above are also significant for this group of cases well beyond the 1 per cent level.

It seems that the fenestration operation benefits particularly the lower speech frequencies on which the threshold measurements for speech depend. Remember that TICD is a direct test of the intelligibility of connected discourse. This is what the patient himself probably uses in his everyday judgment of the benefits of the operation, and the averaged air audiogram, if it includes 2896 or 4096, therefore underestimates the practical gain. The practical gain for speech would be quite well estimated from the average gain for the *three* frequencies, 512, 1024 and 2048.

We must emphasize again that all of the gains for pure tones and for speech that have been considered in this section are early post-operative gains measured usually about three months after operation. The subsequent course of events will be considered in a later report, but inspection of the audiograms indicates that in many cases the hearing for 2896 and 4096 improves significantly between the three-month and the one-year tests. The discrepancy between the gain measured by pure-tone audiometry and by speech tests will probably prove to be less for later tests than for the present series of early post-operative tests.

2. Relation to Use of Microscope.

After the 28th operation in this series the surgeon (TEW) introduced a binocular dissecting microscope as an aid in making the fenestra. The operation can be performed more easily and with greater confidence under the dissecting microscope than under the loupe. Perhaps the fenestra made under the microscope will prove less likely to close and the clinical records show that the patients suffer less from vertigo immediately after the operation, — but we can see no significant improvement in the immediate gains. The mean gain for speech (average of No. 9 and TICD) is 26.0 db ($N = 28$) with the microscope and 25.0 db ($N = 21$) without it. The difference is not significant even at the 5 per cent level, and in any case cannot be differentiated from the possible effects of a general overall improvement in technique or in selection of cases.

The gains in Social Adequacy Index with microscope averaged 43.6 db (N = 31), but only 32.4 db (N = 23) without the microscope. This difference is significant at the 5 per cent level (η = .29); but in view of the constancy of the gain for speech it obviously depends on the selection of cases with better initial discrimination.[3] (Remember that the PB maximum score is not significantly affected by the fenestration operation.)

3. Relation to Type of Bone.

The type of bone encountered in the operation might have some bearing both on the immediate result and on its permanence. The surgeon (TEW) regularly noted whether the bone in each case was brittle (B), hard (H), or soft (S). He also classified the structure of the mastoid bone as sclerotic (Sc), pneumosclerotic (Pn-Sc), or pneumatic (Pn).

No significant differences in the immediate gains from fenestration were found to be associated with either of these

TABLE V.
RELATION OF IMMEDIATE GAIN (3-4 MONTHS) FROM FENESTRATION TO TYPE OF BONE AND STRUCTURE OF MASTOID.

		Gain for Speech: Average of No. 9 and TICD			Improvement in SAI	
Type	N	Mean Gain	η	N	Mean Gain	η
Brittle	2	14.0 db	.29	3	39.0	.06
Hard	24	26.1 db		27	38.0	
Soft	14	25.1 db		14	40.5	
Sc	11	26.2 db	.07	13	40.1	.05
Pn-Sc	12	26.4 db		12	40.0	
Pn	25	25.0 db		28	38.1	

two classifications. The mean gains, both gain for speech and for improvement in social adequacy index, are given in Table V. Tri-serial eta was calculated in each case, but in no case were the differences in the means found significant at the 5 per cent level.

Our data on retention of gains for a year or more (on cases operated with a cartilage stopple) are still too meagre to warrant statistical tests of a possible relationship between the type of bone and the tendency of the fenestra to close.

V. – PREDICTION OF IMMEDIATE GAINS (3-4 MONTHS) FROM CLINICAL DIAGNOSES.

Neither the gain for speech (average of No. 9 and TICD) nor the improvement in SAI is significantly related to the clinical diagnosis in this series. It should be noted, however, that there are only three cases with predominant nerve involvement and no cases that can be classified as "nerve deafness." As a matter of fact, however, the relationship of improvement in SAI is nearly, although not quite, significant at the 5 per cent level. The data are shown in Table VI.

TABLE VI.
RELATION OF IMMEDIATE GAIN (3-4 MONTHS) FROM FENESTRATION TO CLINICAL DIAGNOSIS.

	Gain for Speech: Average of No. 9 and TICD			Improvement in SAI		
Diagnosis	N	Mean Gain	η	N	Mean Gain	η
C	32	26.2 db	.17	35	42.7	.32
CM	15	26.1 db		17	36.4	
MN	3	20.0 db		3	16.5	

VI. – DISCUSSION.

The results of the present series of tests confirm, wherever they are comparable, the results of our first series, — with two exceptions. Points that are confirmed and which can now be accepted with considerable confidence include

a. The test-retest reliability of Test No. 9, measuring hearing loss for speech, is high ($r = .87$). For patients who are candidates for the fenestration operation its standard error of measurement is between 3 and 4 db.

b. With this type of patient the test-retest reliability of the

Social Adequacy Index (for hearing) is about equally high (r = .87). Its standard error of measurement is about five points.

c. Fenestration causes no significant change in the maximum articulation score on the PB test if the test is given at high enough intensity.

d. The maximum PB score varies with clinical diagnosis. It is highest in conductive deafness, slightly lower in conductive deafness with some nerve involvement causing high-tone hearing loss, and still lower if the nerve involvement predominates.

e. The results of pure-tone audiometry (averaged over the speech range) correlate well with tests of hearing loss for speech such as Test No. 9, but such pure-tone audiometry tends to underestimate the gain for speech immediately following fenestration by as much as 5 db.

The most important new findings are

a. The small and doubtful drift (learning effect) encountered with Test No. 9 in the first series is shown not to be significant.

b. The test of Threshold of Intelligibility for Connected Discourse correlates very highly with Test No. 9. Its reliability is almost as high (r = .83), its standard error of measurement is a little greater (by about half a decibel), and it shows no evidence of a learning effect. Nevertheless, Test No. 9 remains our most reliable and generally satisfactory measure of hearing loss for speech.

c. Pure-tone audiometry (average of 512, 1924, 2048 and 2896) shows a significantly lower reliability (r = .59) and larger standard error of measurement (nearly 6 db) than our speech tests.

d. The underestimate by pure-tone audiometry of the gain (for speech) from fenestration is due to a relatively smaller gain at frequency 2896. The separate frequencies 512, 1024, and 2048 each show nearly the same mean gain for fenestration, and this gain is almost identical with the gain for speech

measured by Test No. 9 or by Threshold of Intelligibility for Connected Discourse. The smaller gain at 2896 is not reflected in the threshold measurements for speech, since this frequency is relatively unimportant for the understanding of easy words (the No. 9 list) or for simple everyday connected discourse.

e. The PB score at high intensity is a quite stable measure if it is initially 85 per cent or higher. It is much more variable if it is initially below 85 per cent. In the present series there is definite indication of the expected learning effect, particularly for those whose scores are initially low.

f. The PB score at the intensity level of the Threshold of Intelligibility for Connected Discourse is about 25 per cent in the present series. This is a little higher than our previous result for normal listeners and for another group of hard-of-hearing listeners.

g. The introduction of a dissecting microscope in the operation did not give a greater mean gain for speech. The mean gain for the entire series was 25.5 $\pm$ 2.6 db.

h. The gain for speech and the improvement of the social adequacy index are not significantly related to the type of mastoid bone encountered or to the presence of a moderate amount of nerve deafness.

SUMMARY.

A second series (55 cases of young and middle-aged adults on whom the fenestration operation was performed) was tested (monaurally, both ears) before and a few months after operation by pure-tone air audiometry, by two tests for the threshold of speech (Test No. 9 and Threshold of Intelligibility for Connected Discourse) and for maximum articulation score on PB lists given at high intensity (usually 110 db re 0.0002 dyne per cm^2). The Social Adequacy Index (for hearing) was also calculated.

Data are given on the test-retest reliability, standard error of measurement and intercorrelations of the various tests, and on the gains following fenestration. All of the tests in question show good reliability and all three threshold tests cor-

relate very highly with one another. Learning effects were significant only for the PB words at high intensity, and particularly for patients whose initial scores were below 85 per cent.

Test No. 9 proves to be the most reliable and generally satisfactory test for the threshold of speech for our present type of patient.

The Threshold of Intelligibility for Connected Discourse for this series corresponds to the level at which a patient repeats correctly about 25 per cent of the PB words.

We have confirmed our previous observation that the gain from fenestration estimated by pure-tone audiometry (average of 512, 1024, 2048 and 2896) is less by about 5 db than the gain measured by speech tests. The discrepancy is mainly due to a smaller gain at frequency 2896 as compared with the gains at the lower frequencies.

Other details are summarized in the discussion.

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REPAIR OF THE SEPTAL PERFORATION A RHINOLOGIC PROBLEM. A RHINOPLASTIC APPROACH (AUTHOR'S TECHNIQUE).

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In the field of rhinology, one of the most troublesome conditions to be found is the symptom complex of septal perforation. Its importance varies in most cases in direct proportion to the etiological factors producing it.

The medical literature on the subject is notably sparse, particularly with reference to plastic reparative methods directed at closing the perforation. Apropos of this, Imperatori states, "Plastic operations to turn down a flap of mucosa to cover the perforation are done, but in general the results have not been good enough to warrant their being recommended."

With striking parallelism, W. C. Phillips writes, "In cartilaginous perforations, with healed edges, no treatment should be attempted." Similarly, Coates states, "Small perforations are the most annoying to the patient and can always be converted into larger ones when it is inadvisable or impossible to close them."

SYMPTOMS.

Patients with cartilaginous perforations usually complain of blockage of the nasal passages because of crust formations which accumulate on the margins of the perforations. This complaint is usually associated with atrophic rhinitis. Repeated hemorrhage is a common finding and is often the result of the individual picking at his nose in an attempt to remove

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the crusts. Repeated cauterizations to control the bleeding often results in larger perforations. Respiratory whistling is another symptom, particularly if the perforation is anteriorly placed and is small.

External nasal deformity is a sequela to septal perforation, especially if there has been major destruction or necrosis of the quadrilateral cartilage. The deformity is usually a saddle defect of the septal dorsum or bony dorsum, or both. In the latter case, the usual cause is syphilis.

ETIOLOGICAL FACTORS.

There are fundamentally three varieties of septal perforation: the most prevalent type is the perforation where the cartilaginous portion alone is involved. The second type is the perforation involving the bony portion of the septum. The third type is the perforation involving both the bony and cartilaginous portion of the septum. The etiological factors, associated with the above three types of septal perforations, may be classified as follows:

1. Metabolic: Those associated with diabetes, syphilis, tuberculosis, cancer, blood dyscrasia, as leucemia, and medical conditions, as diphtheria and typhoid fever, arteriosclerosis with recurring hemorrhages and ulceration, scurvy (with hematoma). Here, perforation is the end-result of a catabolic process. This type is often called the spontaneous perforation.

2. Traumatic (those caused by mechanical manipulation with the finger): Knife wounds, accidental injury with pencils or other implements, prizefighting or acts of felony, crash injuries which result in fracture or dislocation with laceration and perforation of the mucous membrane and perichondrium, with or without infection.

3. Surgical: Perforations resulting from operations to correct the deviation of the septum *per se,* or associated with either radical sinus surgery or other operative procedures, often with the rhinoplastic procedure. In small perforations, immediate repair is usually made.

4. Chemical Agents: These are usually associated with compensation diseases, such as sulphuric acid or chromic acid fumes, mercurial poisons, phosphorus burns, or gases, as phosgene.

5. Chronic atrophic sinus disease may also cause septal perforations due to crusting and atrophy of the mucous membrane with exposure of the cartilages and, later, cartilage necrosis, usually associated with secondary infection.

6. Postoperative Causes of Septal Perforation: Improper packing of the nasal cavity following submucous operations. The uneven packing of either plain or vaseline gauze in wads, or circular bundles in the nasal cavity, following submucous resection, in many cases causes undue pressure on the membrane flaps or the nasopalatine (artery) vessels; as a result a postoperative perforation may develop. This may not be apparent for a week or two after the surgical procedure, but it usually follows extreme pressure or it may follow the application of sutures or the removal of suture or trauma in pulling the sutures.

7. Another cause for perforation is postoperative hematoma or post-traumatic hematoma, inadequately or improperly cared for. The mucous membrane and cartilage either break down from circulatory disturbance and pressure or secondary infection with abscess formation.

8. Septal abscess, as a result of nasal infection, or as a result of infection postoperative to submucous resection, often results in septal perforation.

9. Perforations will result from overzealous cauterization to control epistaxis or spontaneous hemorrhage along the septum with chemical agents, the electrocautery or coagulating and dessicating currents. The chemical or thermal process will result in necrosis of tissue.

10. Congenital — only a few cases are on record.

11. Spontaneous perforations *per se.*

PATHOLOGY.

Unrelated to any of the above etiological factors, spontaneous perforations of the septum are associated with spur formation, fracture dislocation, or other malformations of the septal cartilage. There is an alteration of the air currents in the nasal vestibule which gives rise to the collection of mucus and dust on the mucous membranes. The end-result is a drying and irritation of the mucous membrane with the ultimate replacement of the ciliated columnar epithelium with the transitional type. The latter is less resistant than the columnar type of epithelium and is subject to cracking with hemorrhage. Repetition of hemorrhages follows mechanical manipulation with the fingernail or the violent blowing and twisting of the nose with a handkerchief. Following ulceration and perforation of the mucous membrane, the septal cartilage becomes exposed, dries and ulcerates with later perforation. The marginal mucous membrane will gradually close over the cartilage edges by fusion.

TREATMENT.

Medical Treatment: This is given with the use of bland ointments locally, such as yellow oxide of mercury, zinc oxide or boric acid, also, topical application of the mild silver nitrate preparation (1 to 5 per cent) to the edges of the perforation after removal of the crusts. Exposed cartilage or bone is removed with a forceps. Early medical treatment will limit the size of the perforation.

When a constitutional disease is associated with the perforation, as in the case of syphilis, diabetes, tuberculosis, typhoid fever and the blood dyscrasias, then the medical treatment is directed to that end.

Surgical Treatment: It should be the aim of any proposed surgical treatment to restore to the nasal passages the physiologic functional rôle of the septum. Early submucous resection will often correct or alleviate the progress of the pathological process in nonconstitutional spontaneous perforation

cases. Recorded in the literature are four plastic procedures dealing with closure of the septal perforation:

1. Goldstein's operation: This is a plastic flap operation. The free edge of the mucous membrane perforation is pared from the border of the perforation. The perichondrium is elevated and a section of intervening cartilage removed. A mucous membrane flap is then resected from a contiguous portion of the septum and fitted into the space where the cartilage was resected. The flap is held in place by sutures. The backing of this flap, as well as the donor site, must close by healing with granulations. This invites infection and contraction.

2. Chevalier Jackson's procedure is to raise a flap of mucous membrane from the inferior turbinate to close the perforation.

3. F. J. Pratt has proposed the use of a sliding flap of mucous membrane to close the recent perforation or tear.

4. McGivern's method is one whereby the free margins of the perforation are freshened and mucosal flaps liberated around the perforation. A fitted flap of mucous membrane is cut and sutured to wetproof adhesive which is sutured and held in place for four days with iodoform gauze packing.

All the four procedures mentioned above make use of flaps which have mucous membrane on one side and perichondrium or tunica propria on the other. The backing of the flap must close by healing with granulation tissue. This is equally true of the donor site, for no covering is applied to the area from which the flap is borrowed. In consideration of these facts, it is difficult to rule out the possibility of intercurrent infection, as well as contracture of the membrane flap or the membranes proper. With such a possibility, the recurrence of the perforation is likely.

The scope of this paper is to present a surgical technique in the repair of the cartilaginous septal perforation to offset the disadvantages of previous flap methods of repair. The principle underlying the surgical technique proposed is the repair of the perforation by utilization of a double flap repair

so that the continuity of the mucous membrane of the septum is restored on each half, right and left, of the septum. No donor raw areas are created, and inasmuch as the procedure is accomplished through mobilization of the mucous membrane from the cartilaginous and bony portions of the inner nose, adequate surgical approximation can be made without tension.

SURGICAL REPAIR – AUTHOR'S TECHNIQUE.

The procedure is best accomplished in the recumbent position. The nose is packed as for any rhinoplastic procedure. If the case is a postoperative rhinoplasty, then the procedure is planned without an attempt to correct the nasal dorsum unless there is need for secondary correction of the dorsum or tip. The approach is entirely through the interperichondrial tissues. A marginal incision is made within the naris on each side. Through this incision, the mucous membrane is elevated subperichondrially from the lateral alar cartilage and from the inferior side of the upper lateral cartilages. This dissection is then carried towards the septal dorsum and the mucous membrane flap is liberated downward away from the dorsoseptal attachment. The dissection is further completed as far as the caudal or distal end of the septum circumscribing the perforation area on each side. Mobilization of the subperichondrial layer is continued into the region of the perpendicular plate dissecting the mucoperiosteum away from the inferior surface of the nasal bone along the anterior nasal fissure. This completely mobilizes the mucous membrane flap on the lateral and medial or septal wall so that the continuous mucous membrane blanket lies free in the nasal cavity. This mobilization on the septal side is carried down as far as the vomerine ridge or the upper surface of the hard palate. The mobilized flap thus obtained will permit adequate opportunity for direct suture closure of the perforated mucous membrane with silk or catgut after excising the marginal ring of the perforation. If necessary, a form of Z-plasty procedure can be adopted to obtain this end. This procedure is carried out on each side and then the mobilized flaps are replaced as near as possible in their former position, without undue tension.

Suturing of the marginal wound area is then accomplished with interrupted silk sutures or catgut and an intranasal packing of xeroform gauze is lightly packed to maintain the mucous membrane in its anatomical relationship. A single layer of gelfoam, approximately one-half inch by one and one-fourth inches in length, can be placed in a vertical plane

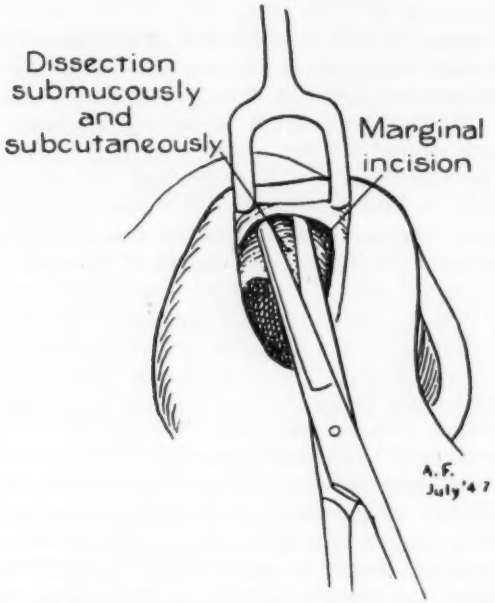

Fig. 1. The beginning of the dissection of the mucoperichondreal and mucoperiosteal flap through marginal incision.

between the mucous membrane or in the interperichondrial space to control postoperative hematoma. A thin strip of preserved or fresh cartilage may be implanted. The procedure outlined has been explained on the basis of a case where no correction of the external nose is necessary or where the correction already done to the external nose has been adequate

and only the question of repair of the septal perforation has been required.

In a case where perforation exists, following any of the above causes, particularly septal perforation following sur-

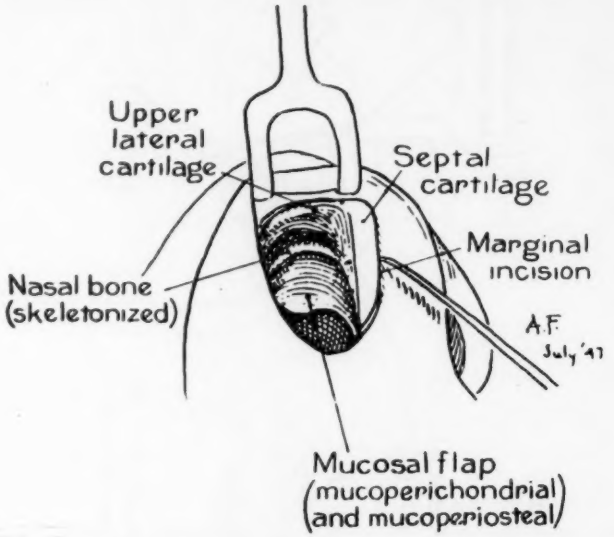

Fig. 2. Mobilization of the mucoperiosteal flap from the nasal bone, septum and upper triangular cartilage is continued until the perforation is exposed completely.

gery, where deformity of the external nose has existed but has not been provided for, then surgical reconstruction or rhinoplasty is contemplated along with the author's method of correction of the septal perforation. This will provide more adequate tissue for repair of the perforation and often will take care of larger perforations. When the mucous membrane is mobilized away from the inferior surface of the upper lateral cartilage and septum, and when the septal and bony dorsum is reduced in height and the distal end of the septal cartilage removed for shortening, much more mucous mem-

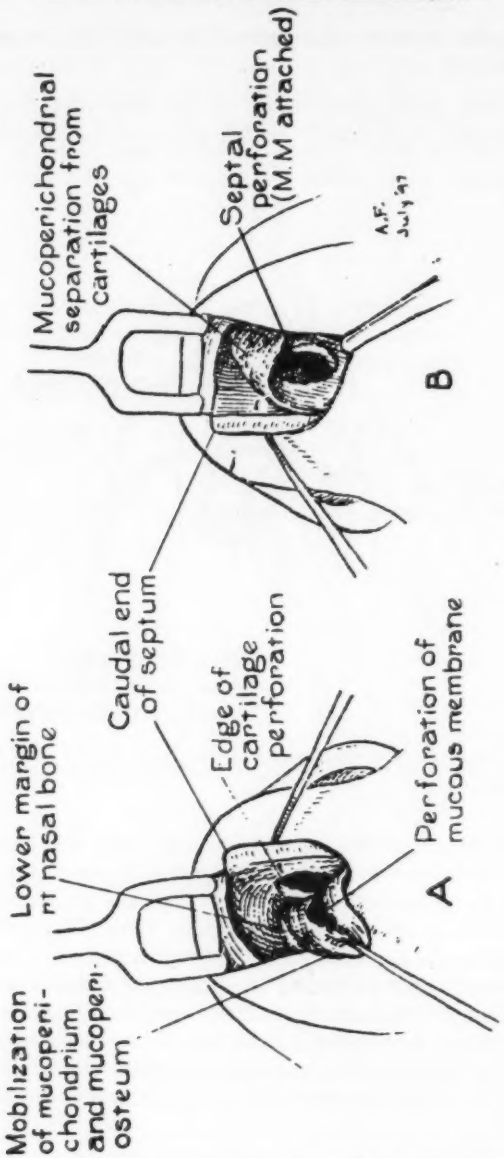

Fig. 3. The completed dissection of the mucosal flap. The perforation is ready for suture.

brane redundancy is obtained and, therefore, larger perforations can be more adequately repaired.

TECHNIQUE DIAGRAMMATIC.

Reference to Fig. 1 in the author's technique shows the dissection being carried out submucously and subcutaneously

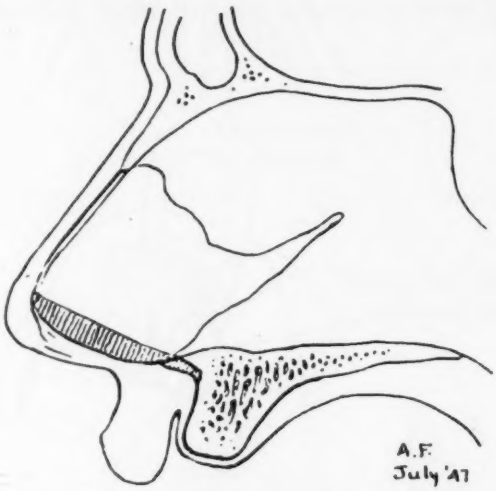

Fig. 4. Shortening of the Septal cartilage provides redundant mucosal tissue for closure of the septal perforation.

through the marginal incision. Fig. 2 reveals the exposure of the caudal end of the septum, the upper lateral cartilage and the right nasal bone have been skeletonized by mobilization of the mucoperiosteum through the marginal incision. This provides a complete skeletonization of the dorsal and medial support of the nose utilized in septal perforation repair. This procedure is adequate also for difficult rhinoplastic correction without perforation. Fig. 3 shows skeletonization of the septal perforation as viewed from the right naris. There has been complete mobilization of the mucoperichondrium and

mucoperiosteum from the right nasal bone. The edge of the perforation in the septal cartilage is clearly visible. The perforation of the mucous membrane is ready for suture. In Fig. 3B the septal perforation is viewed from the left naris and skeletonization of the supportive structure in the left naris is being started. The mucous membrane is still attached about the perforation. Fig. 4 shows that the removal of the caudal end of the septum and part of the anterior edge of the maxillary spine will provide adequate shortening and excess

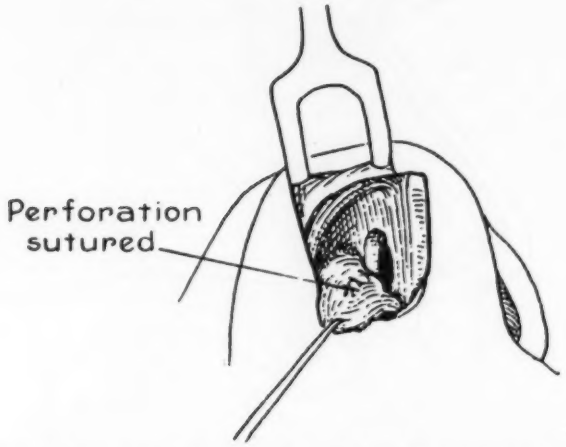

Fig. 5. The right side of the mucosal perforation is closed and the left side is about to be closed.

mucous membrane for adequate closure of the septal perforation. Fig. 5 shows the perforation sutured as viewed from the perichondrial surface.

THE MUCOSAL FLAPS.

It is not necessary that the roof of the mucosa occupy its apical position in the nasal bony fissure between the septum and the lateral and dorsal bony roof. (In saddle noses, the mucosa formerly in the apical position occupies a horizontal

plane.) Further, in rib reconstruction or in bone grafts much of the mucosa is allowed to remain in place by some surgeons. The author mobilizes the mucous membrane freely, however,

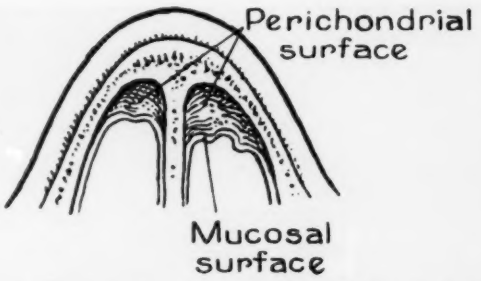

Fig. 6. Diagrammatic representation of mucosal flaps replaced after surgical closure of perforations.

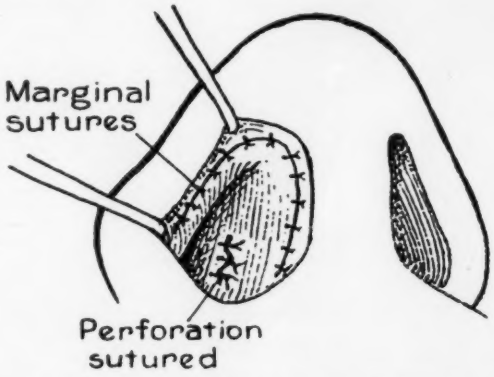

Fig. 7. The appearance of the completed operation.

and after repair repositions the mucous membrane to the contiguous areas of the newly implanted graft. Fig. 6 is diagrammatic and shows position of flaps in nasal cavity.

RESUMÉ.

How to avoid septal perforations: To avoid them is to

repair the rent made in removing spurs and not to regard them as insignificant. These are not true perforations, but they may result in ulceration and, later, perforation when

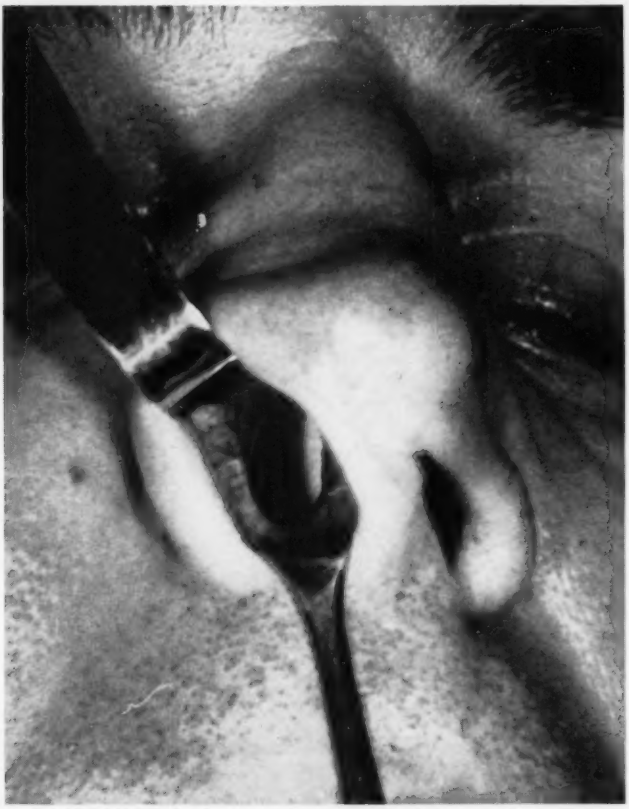

Fig. 8. With retractors in place, a perforation in the nasal septum is photographed but is not clearly discernible.

crusting and postoperative secretion increase. Perforation by puncture on one side needs no repair in the majority of cases, but, nevertheless, it is advantageous to apply a suture rather

than to neglect it. The advantage of developing the mucous membrane blanket flap in rhinoplastic and submucous membrane operations, separate or combined, is:

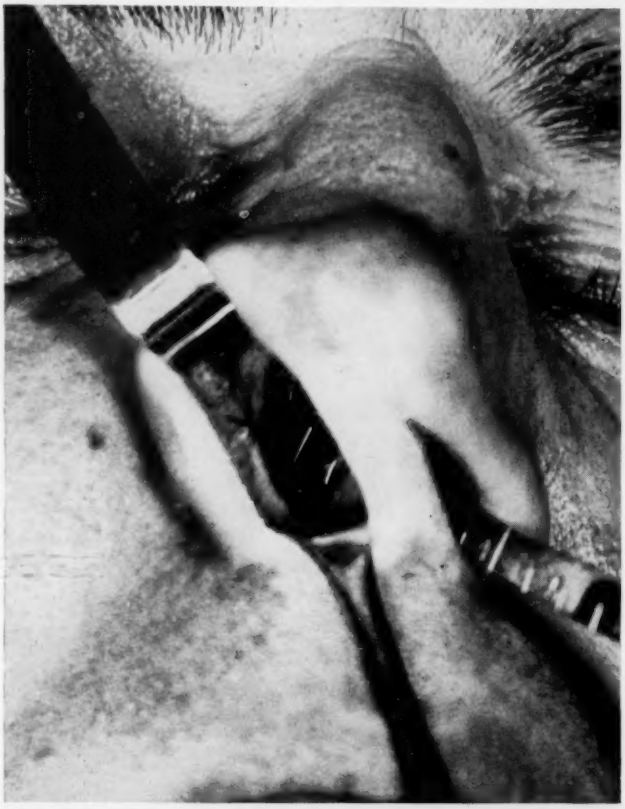

Fig. 9. The same patient. Viewing through the right nares, the perforation is mapped against the background of a metal ruler in the left nares.

1. It prevents injury to the olfactory area or the Schneiderian membrane.

2. The avoidance of injury to the sensory or motor nerves and vascular supply.

3. The prevention of scar formation.

4. The ability to visualize the abnormality of the septum,

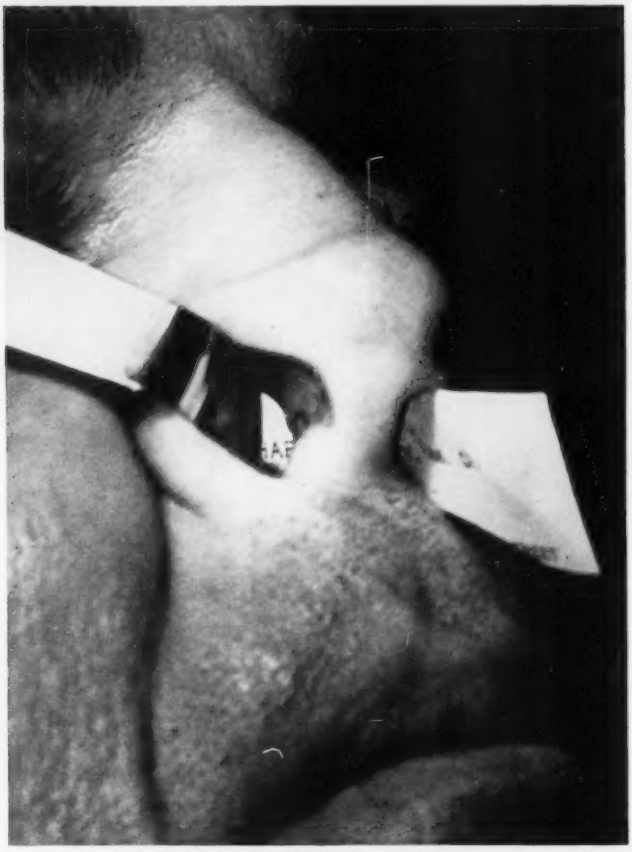

Fig. 10. The same patient. Viewing through the right nares, the perforation is more easily portrayed against the background of a printed card in the left nares.

the vomer and the nasal bone, the knowledge of which presents the opportunity to apply adequate and accurate surgery to these supporting structures.

5. Preservation of the circulation and the innervating nerves results in earlier primary healing.

6. Better visualization is obtained for the necessary surgery.

7. One of the most important factors in the author's technique of mobilization of the mucosal flap is the prevention of atrophic rhinitis and sinusitis by the avoidance of injury to the circulation, thereby eliminating cicatrization of the membrane flaps.

The utilization of the blanket flap of the nasal mucous membrane may be ultimately of value in curing cases of atrophic rhinitis by improving the blood supply in the tunica propria. In addition, it is easier than a previously recommended procedure whereby the medial bony wall of the antra are fractured medialward to prevent atrophic rhinitis.

CONCLUSION.

In review of the literature of the past 15 years, no one has apparently given consideration to the repair or the possibility of repair by the author's method. This procedure may appear difficult; the experienced surgeon, however, will find the procedure gratifying, for it will lead to new concepts and new approaches to the septal membrane and septal cartilage problems, inasmuch as anatomical visualization and interpretations are not restricted.

The sutures used in repair can be made from the mucous membrane or the perichondrial side. In either case, the knot is tied on the mucous membrane side. Hemorrhage is avoided and visualization enhanced. Handling and tear of tissue is reduced to a minimum. Infection is unlikely. Only with this type of approach can linear approximation be made; moreover, any type of approximation is possible, curved or straight, resulting from approximation of torn flaps or even from some form of Z-plasty from remade or secondary flaps. The application of cartilage grafts to the area of the perforation is

more justifiable, inasmuch as a blanket of mucous membrane and perichondrium is recreated to cover the area of perforation on each side.

Restoration of the physiological function of the septum is more likely by utilization of the above method of rhinoplastic repair than with the use of single flaps. Conversely, the complications of procedures utilizing single flaps are: *1.* loss of flaps with recurring perforations often larger than the original; *2.* the development of synechiae with obstructing symptoms; *3.* secondary infection of exposed cartilage at the donor site; *4.* failure of the single flap procedure at operation because of technical difficulties involved; *5.* progress of atrophic rhinitis because of altered circulation.

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POTENTIAL HAZARDS FROM RADIATION TREATMENT OF HYPERTROPHIED LYMPHOID TISSUE IN THE NASOPHARYNX.*†

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Radiation therapy of hypertrophied lymphoid tissue in the nasopharynx, particularly in children and young adults, has become increasingly popular in the last few years.[2,6]. Crowe,[3] who has reported excellent results from this type of treatment, has mentioned the danger of careless use of radium. Radiologists and those familiar with the use of radium have become nervously aware of the growing tendency to give larger and larger doses and to incorporate the method as a routine part of tonsillectomy and adenoidectomy. That radiation of any sort should become a routine is unwise; certainly if this does occur, the results should be checked and rechecked at intervals in order to anticipate any untoward effect. Lack of an immediate reaction should not lull to rest the fears for possible latent reaction. Decision to treat a benign condition with so potent an agent as X-ray, radium or any other radioactive material should be made only after careful consideration of each given case and after weighing the known beneficial results against the unknown dangers which may not appear for as long a time as 10, 20 or more years.

Even today, after 30 years' experience with the use of radium and X-rays, the exact effect and the final result of this form of treatment are unknown. During the pioneering years, radiologists, dermatologists, surgeons and others learned slow-

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ly and painfully something of the good and something of the bad which may result from irradiation of human tissue. Immediate spectacular results often led to tremendous overenthusiasm, and it was not for some years that reports of disastrous effects of treatment to the patient and of overexposure to the operator appeared in the medical literature. Because postradiation damage may be years in developing, it is the purpose of this paper to offer a well meant warning to those new to the use of ionizing radiation as a therapeutic agent, particularly regarding its use in the treatment of benign lesions in the young in whom life expectancy is long. Treatment of malignant lesions in the elderly must of necessity take the chance of late radiation reactions, which if they occur may be considered the lesser of two evils.

Out of a rather large number, a few examples of unfortunate results encountered during the early years of radiation therapy at the Massachusetts General Hospital, the Massachusetts Eye and Ear Infirmary and the Collis P. Huntington Memorial Hospital are given to illustrate that which was discovered by the bitter experience of others in the past.

Case 1 (HH 19-318): In 1919 as an infant, the patient was treated for hemangioma of the forehead, and two years later was considered as a satisfactory result. In 1937, the treated area showed keratoses and ulceration which necessitated plastic surgery.

Case 2 (RB U-536400): A young man, who had a large port wine stain on the right side of his face, was treated by numerous applications of radium in the "early twenties." It is probable that no one area received more than a dose comparable to that now recommended for the nasopharyngeal applicator. The treatments extended over a period of three years, between 1921 and 1924, and although the result was not considered excellent, great improvement in the appearance of the lesion had taken place. In 1947, however, the irradiated area broke down, requiring extensive plastic surgery (see Fig. 1).

The point to be emphasized in both of these instances is that the immediate result was considered cosmetically satisfactory, and it was not until many years later that the actual damaging effect of irradiation was recognized.

Fortunately, malignant degeneration is a rare sequela, but it can occur even when: *1.* the treated area is readily visible; *2.* the patient is followed at regular intervals; and *3.* the surgeon is prompt in removing areas showing radiation reaction.

Case 3 (EC U-609903): This man, a barber, is an example of malignancy resulting from irradiation of a benign lesion. Thirty years ago, because of a chronic dermatitis, he was treated by X-rays, receiving an estimated 3,500 r. to the skin of his hands over a period of years (equivalent to about the dose administered by the nasopharyngeal applicator

when the 12-minute treatment is repeated once). In the last 10 years his hands have shown extensive radiation change, and he has had many surgical procedures for relief of painful ulcerations. Within the past year an epidermoid carcinoma of the skin has developed in the irradiated area.

The present radium applicator for shrinking lymphoid tis-

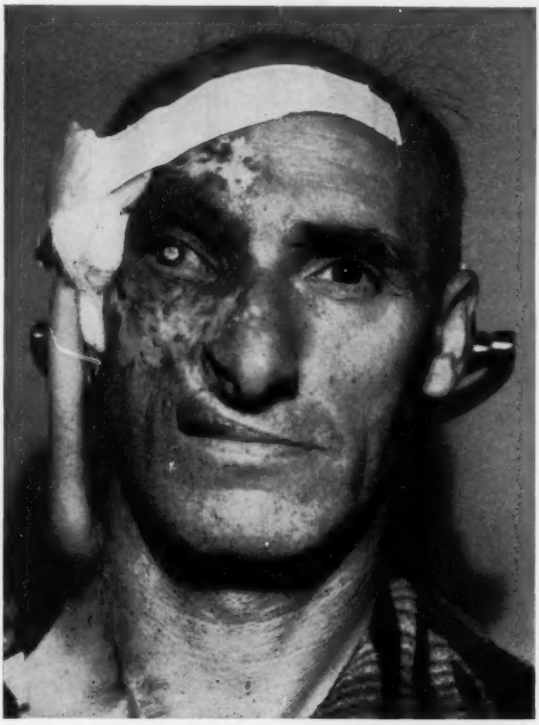

Fig. 1. Case 2. Late radiation changes 24 years after treatment.

sue in the nasopharynx is well known, as is the recommended dosage of 600 mg. minutes, or 10 mg. hours, to each side. Use of this applicator has been thought to be absolutely safe because approximately 80 per cent of the ionizing radiation emergent from it are beta rays (electrons) and 20 per cent

gamma rays. The fact, however, that the effect of gamma rays in tissue is the result of the electrons they produce is frequently ignored. That beta rays and cathode rays of X-ray tubes are electrons and that they are essentially the same, physically and in biologic effect, is likewise either unknown or ignored. Actually, it may be said that the biologic effect of X-rays and gamma rays is, insofar as is known, entirely dependent upon a secondary electron scattering which they incite in the tissue by their absorption. These scattered electrons differ only in their source and energy. In other words, the changes that take place in tissue, whether irradiated by beta, gamma or X-rays, are the same, except that the depth of penetration of electron radiation is less than that of gamma and X-rays. That gamma and X-rays can produce deleterious effects is amply shown in the literature of yesterday and today. To believe that because of their relative inability to penetrate more than a few millimeters, beta and cathode rays are noninjurious to living tissue *is a fallacy*. This was demonstrated by an accident which occurred at the Massachusetts General Hospital in 1944.[7]

Six men were exposed to scattered cathode rays from a supervoltage generator for only a short time in December of that year. The three of the group who were nearest the beam and who received the greatest amount of radiation (an estimated 1,000 to 2,000 r. equivalents, or about the same amount of radiation as that delivered by the nasopharyngeal applicator when applied for 12 minutes) were seriously burned and still carry scars of their injury (see Fig. 2).

The able assistance of Dr. Robley D. Evans, of Massachusetts Institute of Technology, has been requested and given in this study. He believes that measurements with the present monel applicator are highly unsatisfactory and that accurate calculations are well-nigh impossible. On his advice, determination by photographic means of the approximate dosage to the surface and to the depth from various appliances was undertaken. By film blackening, the Roentgen output of both the present nasopharyngeal applicator and the radon applicator previously used in this community was estimated. It

was found that the applicator now in use at the Massachusetts Eye and Ear Infirmary has an output intensity of 150 r./minute at the surface, which diminishes rapidly to about 47.5 r./minute at 2 mm. below the surface; this equals a dose of about 1,800 r. to the surface during a 12-minute treatment (10 mg. hours) and slightly less than 600 r. during the same time

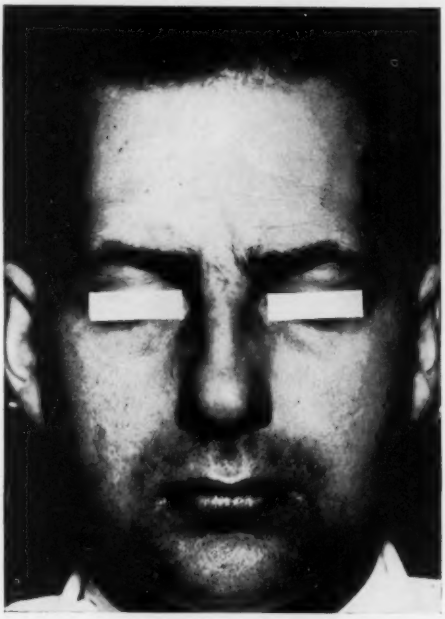

Fig. 2A. 1944. Acute reaction one week after exposure to scattered cathode rays.

2 mm. below the surface of the tissue. The old radon applicator with a wall of steel 0.4 mm. thick gives a somewhat higher output, millicurie for milligram, because the internal absorption is less. By these calculations, this applicator delivers about 185 r./minute at the surface and 75 r./minute at 2 mm. depth. These determinations are considered to be con-

servative in view of the considerably higher figures quoted by Quimby[5] and Boies.[1] Quimby calculated the dose of the nasopharyngeal applicator with 0.3 mm. monel delivered a dose of 1,520 r. at 1 mm. depth per 5.6 mg. hours.

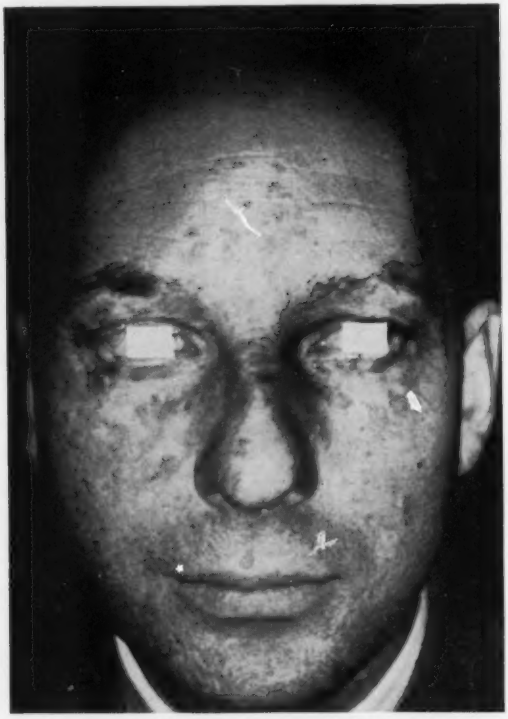

Fig. 2B. Four years later. Note the permanent loss of eyelashes, eyebrows and parts of beard and the extensive changes in the skin.

Because the exact output of this applicator is still indefinite and incalculable, the question arises as to whether the recommended treatment of 12 minutes, often repeated several times, can be considered *invariably* safe. No untoward results from this form of treatment have been published to date, but

the recent development of the procedure eliminates the one actual test of its results; that is, time. Until comparable periods of time have elapsed, the only available test lies in the review of cases in which late radiation damage to tissue other than the nasopharynx followed doses approximately equivalent to those being delivered by the monel applicator. The skin, a site frequently subject to treatment and most easily observed, is best suited for such comparison. Doses of 1,000 to 2,000 r. to the skin as a rule show no unexpected late effects. Skin atrophy usually occurs when a dose of this size is administered in a single setting, and it is well recognized that these atrophic areas are subject to breakdown following trauma or infection. When breakdown occurs, healing is slow and malignant degeneration may follow. Fortunately, untoward results are rare. They are frequent enough, however, to make most radiologists reluctant to give more than an erythema dose in the treatment of a benign lesion.

It is suggested, therefore, that in general this same principle be adhered to in the use of the nasopharyngeal applicator. In other words, in the treatment of the benign lesion of the nasopharynx careful consideration of the seriousness of the lesion should be given before administering more than an erythema dose. What constitutes an erythema dose with this appliance should be determined with a given instrument. If this can be learned in no other way, the operator can apply his applicator to his own arm, using the hairless volar aspect of the forearm as the testing ground, and from the results be governed as to the dosage appropriate to the nasopharynx of his patient.

Acute reactions to amounts of radiation similar to those now recommended for the treatment of hypertrophied lymphoid nasopharyngeal tissue are well known. An erythema of the skin has been seen following as little as 400 mg. minutes of mildly filtered radium, or about 700 r.[8] That these reactions will usually produce some scarring is to be expected. By increasing the dosage, the likelihood of severe sequelae, including atrophy, necrosis and even malignant change, becomes even greater.

It seems reasonable to assume that the mucous membrane of the nasopharynx is probably as vulnerable as the skin. On this premise, therefore, it can be expected that in the years to come a certain number of children now being enthusiastically irradiated by means of the monel applicator in an attempt to reduce hypertrophied lymphoid tissue may develop some degree of damage. If, on the other hand, instead of the recommended dosage, the amount of radiation is limited to an erythema dose, this possibility becomes more remote. It may be that no absolute conclusions can be reached as to the ultimate harmful effect of this method of treatment for another 10 years. In the meantime, the fact should never be forgotten that *it is possible* for ionizing radiation applied to human tissue in the present therapeutic dosage to produce latent severe injury. At the present time the procedure cannot be considered as invariably safe, and its *routine use*, without careful consideration of each individual case, is hazardous.

SUMMARY AND CONCLUSIONS.

In view of the present vogue for treating hypertrophied lymphoid tissue in the nasopharynx with radium, a warning of possible resultant dangers is offered.

Certain unfortunate occurrences encountered by the radiologist or surgeon using radium during the early years in which he was learning the therapeutic possibilities of ionizing radiation have been presented. In these cases, late radiation necrosis had followed treatment of benign lesions and many of the changes in normal epithelium, which were progressive and permanent, had resulted from radiation dosage that was approximately the same as the dose now recommended for use in the nasopharynx for hypertrophy of lymphoid tissue. *The changes were latent, some not appearing for 10 to 20 years after treatment.*

The nasopharynx is a location not ordinarily observed by the patient, and only by chance medical examination will early radiation changes be found. Should such changes develop and progress to radiation necrosis, the nasopharynx

is not a suitable site for plastic surgical repair. Fortunately, breakdown of atrophic irradiated tissue occurs infrequently, and malignant degeneration with even greater rarity; but for the patient in whom either misfortune takes place, the end-result is often worse than the original condition.

With the idea in mind that mistakes of the past may not be repeated in the present, certain precautionary measures are suggested: *1.* that the use of the monel applicator should not be routine; *2.* that for benign lesions in children and young people this treatment should be carried out cautiously and only after careful consideration of each case; *3.* that, except in the selected case, the treatment when given should consist of no more than erythema dose; *4.* that what constitutes an erythema dose should be determined for a given applicator; and *5.* that should no other means of determining the proper dosage for an applicator be available, the operator should estimate it by trial on his own skin.

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STREPTOMYCIN IN OTOLARYNGIC TUBERCULOSIS.*

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In July of 1946, studies of the effect of streptomycin on tuberculosis were initiated at Fitzsimons General Hospital as a component of the cooperative study of the Army, Navy and Veterans Administration. This study group has defined certain definite clinical trends in the response of pulmonary tuberculosis to streptomycin therapy.[1,2,3] Extrapulmonary lesions of all types also have been evaluated under streptomycin therapy, and in most instances with very promising results. Approximately 1,000 cases of tuberculosis of all types have received streptomycin at Fitzsimons General Hospital to date. During this period 85 cases of tracheobronchial tuberculosis and 57 other otolaryngic lesions of tuberculous nature have been available for study. It is proposed to report upon and evaluate the cases from this group which were selected for streptomycin therapy.

INCIDENCE OF OTOLARYNGIC TUBERCULOSIS.

For the past several years there has been a decided decrease in both the incidence and severity of tuberculosis of the upper respiratory tract and its environs. Most phthisiologists feel that this reduction in the incidence and severity of upper respiratory tract tuberculosis is explained by such factors as elimination of bovine tuberculosis through public health measures, earlier diagnosis, isolation and treatment of the patient, and the therapeutic procedure of collapse therapy. More recently, the general use of sulfonamides and antibiotic therapy has done much to mitigate the effects of secondary pyo-

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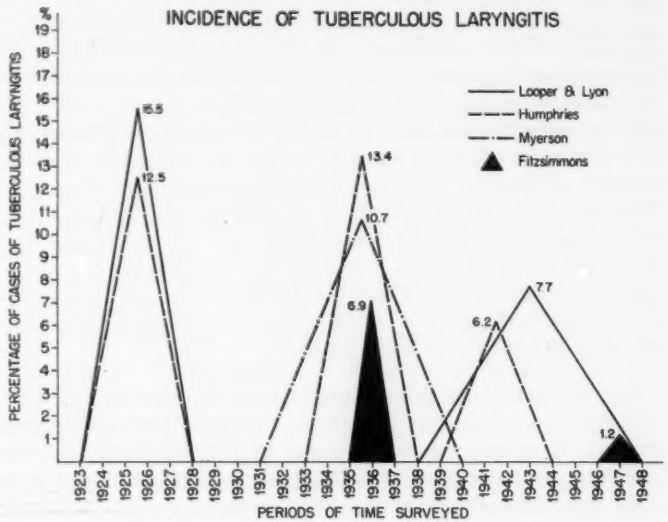
genic infection in tuberculous lesions. This has had a beneficial effect on the tuberculous lesion and lessened the degree of severity in the average patient.

Some of the recent reports reflect trends in the incidence of tuberculous laryngitis. Fig. 1 has been prepared from the



reports of Humphries,[4] Myerson,[5] Looper and Lyon,[6] and from a survey conducted at Fitzsimons General Hospital, which is illustrated in Table 1.

The period from July, 1946, to July, 1948, represents the two-year interval during which streptomycin became available. During this interval certain cases of pulmonary and extrapulmonary tuberculosis were selected to receive streptomycin therapy. Not all of the cases recorded in Table 1 received the drug, and some were terminal cases which provided little clinical information.

The rather remarkable decrease in the incidence of oto-

TABLE 1. INCIDENCE OF OTOLARYNGIC TUBERCULOSIS AT FITZSIMONS GENERAL HOSPITAL.

	Tbc. Patients	No. Tbc. Laryngitis	% Tbc. Laryngitis	No. Tbc. Otitis Med. Mastoiditis	% Tbc. Otitis Media	No. Tbc. Pharynx	% Tbc. Pharynx
July, 1935 July, 1937	4,623	317	6.9	46	1	4	.25
July, 1946 July, 1948	4,000	48	1.2	8	.2	1	.025

laryngic tuberculosis, and particularly tuberculous laryngitis (1.2 per cent of 4,000 cases), seems to parallel a lessened severity of lesions. Extensive inflammatory and edematous reactions, such as characterized tuberculous laryngitides in so many cases observed ten or five years ago, are to be seen only in a few terminal cases. The typical turban hood type of epiglottic reaction and rapidly destructive lesions, in particular, appear infrequently. Progression of lesions to cicatrization and deformity is seldom seen.

Before attributing the recent remarkable decrease in tuberculous upper respiratory infections to the utilization of streptomycin, it should be remembered that early diagnosis and other therapeutic measures which include antibiotics and judicious uses of collapse therapy have already done much to minimize these complications. Streptomycin may be regarded, however, as a therapeutic measure which is competent to halt the progression of pulmonary tuberculosis and the consequent appearance of extrapulmonary lesions of the upper respiratory tract. Consideration of the more specific response of otolaryngic lesions to such therapy will require continuing study.

EFFECT OF STREPTOMYCIN ON TUBERCULOUS LESIONS.

Much information on the effect of streptomycin on tuberculous lesions is derived from the many clinical reports of the effects of this drug on pulmonary tuberculosis. In general, the drug appears to be particularly effective in fresh exudative lesions where vascularization permits ready access of the

drug to the tuberculous organism. As lesions become productive of scar tissue or progress to necrosis, caseation or cavitation, accessibility of the organism is lessened and effectiveness of the drug is consequently diminished.

The extensions of pulmonary infection to the respiratory mucosa of the tracheobronchial tree, the larynx, nasal cavity or ear are largely those of clinical exudative lesions with relatively good vascularization, relatively thin submucosal stroma and superficial situation. There would then seem to be reasonable histopathologic basis for anticipation of good clinical response in lesions of these areas. An exception reserved for later comment concerns the more remote structure of the mastoid cells.

As with other antibiotics, development of drug resistance is encountered in the treatment of tuberculosis with streptomycin.* Where tubercle bacilli have persisted throughout the long courses of streptomycin, slightly more than half have become resistant. With the short course of streptomycin (1 gm. for 28 days) no cases to date have shown bacterial resistance to streptomycin. It appears, however, that this problem is no simple one. There are frequent instances of good clinical response of otolaryngic lesions to streptomycin in the presence of streptomycin resistant organisms from the sputum. Pending further bacteriologic investigations, it is not advisable to withhold medication on the basis of this laboratory finding of streptomycin resistance without clinical trial.

TUBERCULOUS LARYNGITIS.

Twenty-seven cases of tuberculous laryngitis have been selected for evaluation. Six of these cases have been prepared in detail by Greif and Gould[7] and are included with additional follow-up studies.

The selection of cases has been limited to those meeting definite criteria for clinical diagnosis. The characteristic appearance of tuberculous laryngitis persisting for two to three

*Criterion for resistance was established as growth on 10 or more micrograms of streptomycin per ml.

weeks on repeated examination by two or more experienced observers was accepted as diagnostic. In 15 cases, biopsy confirmed clinical impressions, while autopsy studies were positive in two additional cases in which biopsy was not done.

Method of Treatment: Administration of streptomycin for the cases in this report has been by the intramuscular route. Aerosolized streptomycin proved unsatisfactory early in our investigations and was soon abandoned as a means of administration. In three cases, failure of response to aerosolized streptomycin was followed by prompt response to intramuscular injection. One case responding to aerosolized streptomycin relapsed after a few weeks and subsequently responded satisfactorily to intramuscular injections. Combined therapy with aerosolized and intramuscular streptomycin demonstrated no superiority over intramuscular therapy alone and was discontinued as a method of therapy.

The dosage of streptomycin employed prior to June, 1947, was 1.8 gm. per day (divided into 0.3 gm. every four hours). After this date a dosage of 1 gm. per day (divided into 0.5 gm. twice a day) was uniformly adopted in conformity with the project outline of the Army, Navy and Veterans Administration Committee.[3] Treatment was continued 90 to 120 days with the exception of a few cases in which drug sensitivity developed or there was (fatal) termination.

Clinical observations of the response of laryngeal lesions to 1.8 gm. of streptomycin per day as contrasted with 1 gm. per day dosage have failed to show significant difference in the number of cures or rapidity of response. Recently we have treated patients with short courses of streptomycin, consisting of 1 gm. daily for 28 days, and the results have been most gratifying.

Development of Tuberculous Laryngitis in Streptomycin-Treated Patients: Among approximately 1,000 patients receiving streptomycin for pulmonary tuberculosis, only one case developed tuberculous laryngitis during active treatment. This laryngeal lesion appeared 90 days after streptomycin therapy was started and coincided with the demonstration of

streptomycin resistance. Three cases of tuberculous laryngitis, which are included in this report, developed tuberculous laryngitis subsequent to streptomycin therapy. In each of the cases a resistant organism was demonstrated. There were no cases which developed following streptomycin therapy in which resistance of the organism to streptomycin had not developed.

Response of Tuberculous Laryngitis to Streptomycin Therapy: In patients responding favorably to streptomycin therapy the results were immediate and gratifying from a symptomatic standpoint. Pain and dysphagia disappeared within a week, at which time laryngeal examination revealed less inflammatory reaction. Hoarseness abated and the larynx resumed normal appearance in as short a period as three weeks. Seldom was more than six to eight weeks required for clinical cure. Results are summarized in Table 2.

TABLE 2.

Clinically cured			19
Streptomycin sensitive			17
Previous streptomycin therapy		3	
No previous streptomycin therapy		14	
Streptomycin resistant			2
Previous streptomycin therapy		2	
Resistant in previous therapy		1	
Resistance during current therapy		1	
Improved			3
Streptomycin sensitive			0
Streptomycin resistant			3
Resistance in previous therapy		1	
Resistance during current therapy		2	
Unimproved			5
Streptomycin sensitive			0
Streptomycin resistant			5
Resistance in previous therapy		1	
Resistance during current therapy		4	
Total cases treated			27

From the table, it is evident that the results of treatment of tuberculous laryngitis with nonresistant organisms was uniformly good in the 17 cases of this type cited. On the other

hand, presence of resistant organisms did not preclude healing of the lesion in the two instances, and overall results (which include both resistant and nonresistant organisms). approximate 80 per cent. Not indicated by the table was a tendency for streptomycin to ameliorate the symptoms of tuberculous laryngitis in those cases proceeding to death from extensive pulmonary tuberculosis and in which no observable change in laryngeal appearance was recorded. The need for topical anesthetic applications, cauterization or superior laryngeal nerve injection was practically eliminated.

Relationship of Laryngitis to Pulmonary Lesions: Although the preponderance of laryngeal lesions developed in far advanced tuberculosis with cavitation or lesions producing large numbers of tubercle bacilli, the response of the laryngeal lesion did not always parallel that of the pulmonary lesion. In the two cases cited in Table 2 in which laryngitis developed after previous treatment, with resultant streptomycin resistant organisms, one proceeded to death within three months with no evidence of tuberculous laryngitis on histologic examination of autopsy material; the other case was clinically cured of his laryngitis after six months' follow-up, while the far advanced pulmonary lesion remained unchanged. In general, however, improvement of pulmonary and laryngeal lesions was concomitant, while those cases dying of far advanced streptomycin resistant tuberculosis represented the majority of the laryngeal lesions which were unimproved.

Recurrence of Laryngitis After Termination of Therapy: Of the 18 cases reported as clinically cured, 13 have been available for re-examination over intervals ranging from four to 16 months without evidence of recurrence.

Two cases which might be considered recurrent were encountered. In one instance of aerosol therapy alone, recurrence was noted after initial improvement. Intramuscular therapy was then instituted and the lesion cleared.

The other case was interesting with regard to the histopathologic findings. This patient demonstrated persisting hoarseness after apparent healing. Direct laryngoscopy ex-

posed a nodular mass in the ventricle which was excised and proved to be a rather firm tuberculomatous lesion. After a second course of streptomycin, complete healing ensued without evidence of recurrence after several months.

As cited in the second report of the Army, Navy and Veterans Administration study,[3] statistical indication to date suggests that an 8 to 13 per cent incidence of relapse may be anticipated in all types of tuberculous lesions under streptomycin therapy.

TRACHEOBRONCHIAL TUBERCULOSIS.

Evaluation of the cases of tracheobronchial tuberculosis observed in the endoscopic clinic at Fitzsimons General Hospital[8] and summarized in the Army, Navy and Veterans Administration Report,[2,3] as well as the recent reports of Brewer and Bogen[9] and O'Keefe,[10] suggest a close similarity in the efficacy of streptomycin in laryngeal and tracheobronchial lesions. Healing is to be anticipated in 80 to 90 per cent of all types of tracheobronchial disease and ordinarily is complete in six to 16 weeks. Of greatest clinical importance is an arrest of the tendency for endobronchial disease to progress to fibrostenosis of the bronchi with the resultant serious complications of bronchial obstruction. A policy of endoscopic examination and correction of endobronchial lesions prior to collapse therapy permits a more rational application of the various procedures for collapsing the lung. In many cases elimination of endobronchial tuberculosis permits collapse therapy which would otherwise be contraindicated.

OTITIS MEDIA AND MASTOIDITIS.

Seven cases of otitic tuberculosis were treated with streptomycin. The technique of treatment was that previously described for laryngeal tuberculosis. All were bacteriologically positive for tubercle bacilli. Table 3 summarizes clinical experience with these lesions.

The few otitic lesions available for study responded well where streptomycin sensitive organs were present. This was

TABLE 3.

Otitis media	5
Streptomycin sensitive	3
Healed	3
Streptomycin resistant	2
Unimproved	2
Mastoiditis, postmastoidectomy	1
Streptomycin sensitive	1
Healed	1
Mastoiditis, bilateral, facial palsy, extradural abscess	1
Streptomycin sensitive	1
Healed	1*

true in spite of extensive pulsonary lesions which were present in four of these cases of otitis media. Most impressive was the replacement of extensive loss of the tympanic membrane and immediate arrest of deafness. In two cases, gain of hearing function was noted, despite the destructive nature of the tuberculous process.

The two mastoid lesions provided interesting analogies to the ineffectiveness of streptomycin in the presence of caseating or fibrotic lesions of the lungs.

In the cases listed as responding satisfactorily as postmastoidectomy mastoiditis, the patient had had no improvement on streptomyin therapy elsewhere. After therapy was discontinued, a mastoidectomy was performed at a second institution without streptomycin therapy. On admission at Fitzsimons General Hospital six months later, there was persisting purulency from the external canal and a postauricular fistula. Response to streptomycin was prompt with closure of the fistula, cessation of discharge and restoration of an intact tympanic membrane.

The case with bilateral tuberculous mastoiditis evidenced progression of mastoid involvement to facial palsy and extradural abscess prior to determination that the nature of the infection was tuberculous. Response to surgery and streptomycin therapy was gratifying, both in complete cessation of discharge and return of useful hearing from a severe grade of deafness.

*Following bilateral mastoid surgery and streptomycin therapy.

These two cases may well indicate that further studies will lead to modification of the "hands off" policy in dealing with tuberculous mastoiditis in favor of combined surgical drainage and streptomycin therapy.

LABYRINTHINE TOXICITY.

Labyrinthine symptomatology as a result of streptomycin therapy has been observed in various dosages, and certain group studies have been made.

In evaluating labyrinthine manifestations, various clinical procedures have been routinely used. General neurological examination with particular attention to the Romberg test, observation of positional or spontaneous nystagmus, and caloric and galvanic labyrinthine tests have been used.

The caloric test used is a modification of the Kobrak test in which only 1 cc. of ice water is used and the head tipped back 60 degrees to maximal position. The time to the appearance of nystagmus, recorded as the latent time, is compared to the duration of nystagmus. A normal ratio is regarded as 1:3 to 1:5. A ratio between 1:1 and 1:2 is considered mild depression. Ratio of 1:1 or failure of response after one minute is regarded as severe depression, and further testing is not attempted, since such patients seem clinically to have no useful labyrinthine function left.

We have little to add to the excellent earlier descriptions of Brown and Hinshaw,[11] Fowler and Seligman[12] and subsequent observations of these and other investigators as to the clinical signs and symptoms seen in streptomycin toxicity of the labyrinth. Associated with symmetrical depression of both labyrinths and vertigo aggravated by motion, this toxicity effect is without positional or spontaneous nystagmus, and lacks directional falling reactions. Aggravation of symptoms while reading led us to special investigation of the eye muscle nuclei by optokinetic nystagmus testing, study of phorias, fusion and accommodation without evidence of abnormalities.

An incidence of 90 per cent labyrinthine depression begin-

ning around the fifth week of therapy was encountered in our earlier 2 gm. and 3 gm. per day dosages of streptomycin. It was felt that under these dosages almost all labyrinths would be damaged by continuous therapy. Recovery, as noted by Fowler, proceeded by adaptation of equilibration rather than by labyrinthine recovery. In a follow-up of some 34 available cases, ranging from four months to one year, an isolated example of subsequent recovery of labyrinthine response was noted.

With reduction of dosage of streptomycin to 1 gm. per day in a group of 50, the incidence of labyrinthine disorder dropped to only 4 per cent which were considered severe by labyrinthine testing. Lesser grades of depression were observed in an additional 24 per cent. Most encouraging, however, was the fact that a survey conducted four to five months after cessation of therapy demonstrated return of caloric reactions to normal in better than half of the mildly depressed cases. Indications were that additional cases would recover eventually.

Of interest among the mildly depressed group were several who developed depression of caloric reaction without vertigo. Apparently adaptation was keeping pace with labyrinthine damage. In other cases, patients who were asymptomatic in the quieter confines of the hospital complained of vertigo and ocular difficulties after resuming normal activities on the outside. Driving cars over bumpy roads or difficulty in reading road signs while in motion were usual complaints. In a similar manner, patients immobilized in orthopedic appliances sometimes noted vertigo for the first time when permitted greater latitude of motion.

In two recent experimental groups of 50 patients, one receiving 2 gm. and the other 3 gm. of streptomycin per day at three-day intervals, repeated caloric tests over a period of four weeks to date have failed to show evidence of labyrinthine damage. A group of cases in which the dosage of streptomycin has been reduced to 0.5 gm. per day is also under study by the combined Army, Navy and Veterans Administration

group. These techniques appear promising with regard to labyrinthine disability providing such dosage is therapeutically efficacious.

Regarding loss of cochlear function, we have observed only one case of deafness attributable to streptomycin therapy. This was in a case of tuberculous meningitis. Variations in audiometric studies before and after therapy were considered within normal limits on 100 patients selected at random. Although there may have been an increase in the incidence of tinnitus, this very common symptoms of essentially, normal individuals was not particularly impressive.

SUMMARY.

1. A summary of clinical experience in streptomycin treatment of otolaryngic tuberculosis over the past two years at Fitzsimons General Hospital is presented.

2. The incidence of otolaryngic tuberculosis has been rapidly decreasing in recent years due to improved therapeutic measures. Graphic representation of the trend in laryngeal tuberculosis is presented, showing a drop in incidence to 1.2 per cent of tuberculous cases at Fitzsimons General Hospital during the two years in which streptomycin was employed in treatment of pulmonary disease.

3. Excellent response of tuberculous laryngeal lesions to either 2 gm. or 1 gm. per day dosage of streptomycin is recorded. Clinical cures were obtained in 18 out of 28 cases, with improvement in five others. Failures were related largely to streptomycin resistance of the organisms. Amelioration of symptoms was noted in cases which did not respond with clinical healing. There appeared to be slight tendency for recurrence after healing in the group of cases presented. The results of treatment of tracheobronchial tuberculosis approximate those of laryngeal lesions.

4. Otitis media responded with rapid healing in three cases and poorly in two streptomycin resistant cases. Mastoiditis was controlled by surgery supplemented by streptomycin therapy in two cases.

5. Labyrinthine toxicity, which was severe in 90 per cent of patients on 3 gm. and 2 gm. per day dosage of streptomycin, was reduced to 4 per cent incidence of severe grade of labyrinthine reaction when on 1 gm. per day dosage. Milder grades of labyrinthine depression, occurring in 24 per cent on the 1 gm. per day dosage, showed recovery of labyrinthine responses in over half the cases.

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MENINGITIS FOLLOWING NASAL POLYPECTOMY.*

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The removal of nasal polyps is usually accomplished without complication, but the possibility of serious complications is well recognized.

The mucosa of the ethmoid sinuses is, from its histologic characteristics, more subject to the formation of polyps than is the mucosa of other parts of the nose. This fact has double significance in the spread of infection to the cranial cavity after removal of polyps: first, the anatomic proximity of the ethmoid capsule to the floor of the anterior cranial fossa, and second, the retention of infection in the ethmoid cell whose ostium is blocked by a polyp.

One of the outstanding pathologic changes associated with nasal polyps is the absorption of the bony walls of the ethmoid sinuses and the turbinates as a result of pressure. The intercellular septa of the ethmoid become almost nonexistent in severe cases of polyposis. The middle turbinate may be absorbed, and the lamina papyracea absorbed and displaced. The same process occurring in the roof of the ethmoid exposes the dura to direct contact with infected material when the ethmoid is exenterated and polypoid mucosa is removed.

The small incidence of intracranial infection following removal of nasal polyps attests the skill of the rhinologic surgeon in performing this delicate procedure. Due to the small incidence of complications, few are seen by any one surgeon and consequently only a small proportion of the cases which occur are reported in the medical literature.

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Loeb,[1] in 1922, sent questionnaires to 5,000 practicing otolaryngologists throughout this country in search for unreported fatalities, exclusive of deaths due to anesthesia, resulting from operations on the nose and throat. Two hundred and fifty surgeons reported 332 fatalities. Meningitis was the cause of death following 125 intranasal operations, of which 13 followed removal of polyps.

In 1916, Dabney[2] reported nine deaths following polypectomy. Two resulted from meningitis, two from brain abscess, one from septicemia, and one from latent meningitis. In the last case there was a preoperative history of headache and fever. The cause of death was not given in the remaining three cases. Dabney expressed wonder that more such deaths had not occurred, since polyps are so often superficial to periosteal or bone disease and are usually removed by avulsion. This must set free infection in the presence of new areas for absorption in the middle and superior straits of the nose, a region which is always dangerous. He quoted a case of Hayen, who packed a nose with gauze soaked in perchloride of iron to stop epistaxis and saw his patient die some hours later of meningitis. Necropsy showed the inflammatory process plainly, and also the path of infection, which proved that the olfactory nerve fibres transmit infection through the cribriform plate to the meninges. The whole length of the olfactory sulcus was stained brown and this discoloration continued throughout the sheaths of the olfactory nerves to the meninges.

Davis,[3] in 1939, stated that he knew of six recorded cases of injury to the roof of the nose and had seen seven patients in consultation. The injuries occurred during removal of nasal polyps with forceps or during operations for sinus suppuration in which forceps were used for the removal of the ethmoid cells. All injuries occurred in the region of the posterior ethmoid cells when forceps were directed upward and backward. He pointed out that such injuries may be avoided by directing forceps parallel to the roof of the nose.

Imperatori,[4] quoting Turner and Reynolds,[5] stated that

there are three pathways for extension of infection from the nose to the meninges: *1.* by way of the venous blood stream (48 per cent), *2.* direct extension through the bone (43 per cent), and *3.* by the olfactory perineural sheaths (9 per cent). Imperatori's series consisted of 17 cases of rhinogenic meningitis. In seven the meningitis complicated paranasal sinus disease; in seven it followed intranasal operations on the sinuses, in two, submucous resection of the nasal septum, and in one a cavernous sinus thrombosis secondary to a nasal furuncle.

Hagerup[6] reported 33 cases of spontaneous intracranial complications of rhinogenous origin. In two the complications followed operative trauma, and in one, cerebral injury with involvement of the frontal sinus.

Eagleton[7] warned of the danger of operating on patients who gave a history of headache or vomiting, because in his experience the meningitis following intranasal surgery is only a recrudescence of a former intrameningeal infection.

Brunner[8] wrote that intracranial complications following removal of nasal polyps are not uncommon. He stated that meningitis subsequent to intranasal surgery may be caused by *1.* fracture of the cribriform plate, *2.* infection of the sheaths of the olfactory bundles, and *3.* acute infection or exacerbation of a chronic sinus infection. He stated that when polyps are removed with a conchotome from the roof of the ethmoid sinuses or from the superior meatus a fracture of the cribriform plate is the usual cause. When polyps are removed from the middle meatus with the cold snare, a chronic sinus infection may flare up, particularly if the endonasal operation did not achieve satisfactory drainage of the involved sinus. Pus retained in the sinus in turn causes the chronic sinus infection to spread toward the meninges. He cited a case in which meningitis and death followed extraction of polyps. Evidence at necropsy indicated that meningitis was impending before removal of the polyps. The surgical procedure caused acute exacerbation of the chronic sinus infection. Owing to the exacerbation of the chronic infection, the infection spread

rapidly into the nasal mucosa and sheaths of the olfactory bundles on both sides. There was no fracture of the cribriform plate.

The rôle of the network of lymph capillaries which accompany the meningeal sheaths in the olfactory mucosa in carrying infection to the intracranial space has aroused much contradictory comment. Brunner[8] stated that next to the meningeal sheaths of the olfactory nerves is a network of lymph capillaries which, in the newborn infant, is believed connected with the subarachnoid space. The communication is provided by small canaliculi which pass through the foramina of the cribriform plate with the filaments of the olfactory nerve. This network is not in communication with the lymphatics of the respiratory portion of the nose. He has traced lymphatics running in the mucosa of the lateral nasal wall from the olfactory area up to the cribriform plate, but could not trace them to the subarachnoid space. Rosenvold[9] cited the contradictory literature on this subject. Zwillinger[10] demonstrated this connection between the subarachnoid space and the lymphatics of the olfactory portion of the nasal mucosa by placing dye in the subarachnoid space and later finding it in the olfactory mucosa. Turner and Reynolds[5] expressed the opinion that this connection does not exist. They did recognize, however, that peripheral lymphatic vessels take origin in the inner layer of the dura mater. Rosenvold[9] stated that the clinical significance of this avenue of infection, if it exists, is greatly overestimated. He demonstrated that in rabbits the spread of infection from the nasal septum to the intradural contents favored a vascular (thrombophlebitic) route.

Since intracranial infection after nasal polypectomy may be caused by the flare-up of chronic sinusitis, the pathways for extension of infection from the sinuses to the meninges are important.

To Hajek[11] is given credit for the first detailed study of these pathways. These he outlined as follows: *1.* direct extension through contiguous structures with macroscopic evidence of destruction of the mucous membrane and bony walls may

occur. An intradural abscess then forms on the cerebral side of the bone and may give rise to meningitis, cerebral abscess or sinus thrombosis. *2.* Regional metastasis in which the bone bordering the cranial cavity is not destroyed may occur. The discoloration of the dura and the underlying bone is the only indication that the process might have penetrated this designated area. Hajek[11] concluded that this extension occurs via a perforating vein. *3.* Infection may spread by continuity through macroscopically intact bone. To illustrate this type, Hajek cited a case of Ortman in which there was microscopic evidence in macroscopically intact bone of infection to the meninges by direct continuity through the bone.

Kramer and Som[12] removed the ethmoid and sphenoid sinuses and upper part of the nasal chambers *en bloc* at necropsy in 50 cases of meningitis and studied serial sections microscopically. They stated that this is the only way of ascertaining the source of the meningeal infection. They said that the finding of suppuration in a paranasal sinus or of meningeal exudate at the base of the brain does not prove the rhinogenous source of the infection. Further, macroscopic evidence of extension through bone is so rarely seen (in three out of the 50 cases in their series) that only serial section and microscopic study may be depended upon to determine the rhinogenous source of infection.

They listed the pathways of infection proved or suggested prior to their study as: *1.* direct extension through the bony wall of a diseased sinus as a result of osteomyelitis (Turner and Reynolds, Hesse, Kramer and Som); *2.* lymphatic extension via perineural lymph sheaths (Jacobsgaard, Turner and Reynolds); *3.* venous spread via perforating vessels to the cavernous sinus (Hajek); *4.* congenital dehiscences; and *5.* metastasis during sepsis. Kramer and Som demonstrated all these except metastasis due to sepsis, and added two others.

Before we list the pathways demonstrated by Kramer and Som, we shall refer briefly to the pertinent anatomy as given by these authors.[12] The mucosa of the sphenoid and ethmoid sinuses is separated from the cranial cavity by a common bony

wall. This wall varies in thickness and in structure. The roof of the ethmoid sinus is composed almost exclusively of compact lamellar bone as are the roofs and walls of the sphenoid sinus when the sinus is well pneumatized. Just as frequently, however, cancellous, spongy bone separates the sphenoid sinus from the sella turcica. The floor of the sphenoid sinus is formed by cancellous bone containing marrow. In compact bone, venules from the mucoperiosteum of the sphenoid sinus pierce the bone and communicate with one of the larger cranial sinuses or with dural vessels. These vessels are surrounded by perivascular spaces which are probably lymph channels that communicate with those within the cranium.

In cancellous bone, the deep layer of the mucoperiosteum sends at intervals microscopic digitals into the bone substance. These reach into the marrow spaces. From the marrow spaces diploic veins communicate with the cavernous sinus either directly or through tributaries. These periosteal extensions and perivascular spaces furnish avenues for spread of infection from mucosa to bone to cranial cavity.

The pathways or methods of extension demonstrated by Kramer and Som[12] are as follows:

1. Osteitis and osteomyelitis may permit spread through the bone. Osteitis may cause necrosis of lamellar bone which lacks marrow. On microscopic examination in such cases, loss of lamination, disappearance of bone corpuscles and peripheral erosion are evident. This state may lead to gross dehiscence with direct approximation of the sinus and the dura. Osteomyelitis of cancellous bone occurs *1.* by direct spread into bone from the inflamed mucosa along pre-existing mucoperiosteal extensions, *2.* by formation of a subperiosteal abscess with direct exposure of bone to suppuration, and *3.* by extension of microscopic purulent phlebitis from the mucosa into the spaces in the marrow. Extension to the bone occurred after an acute exacerbation of the chronic infection. In most cases extension is of this type.

2. In one case in their series of 50 cases spread was via the

perineural lymph spaces. They stated that perineural infection around the larger radicles above the cribriform plate is frequently seen in suppurative meningitis. This finding, however, does not imply spread from the nose by perineural pathways unless the perineural sheaths of small radicles of the olfactory nerve below the cribriform plate are also infected. When they are not, the large radicles above the cribriform plate are probably filled with purulent material by seepage from the affected subarachnoid space into perineural sheaths as a result of high cerebrospinal fluid pressure.

3. Perivascular lymph channels also were found to be a pathway for infection. Acute flare-up of a submucosal abscess causes a break through the capsule of the abscess to the subperiosteal space. Infection is spread from this point by the perivascular lymph channels of the perforating vessels through the entire thickness of the bone. An extradural abscess is formed at this site.

4. Vascular spread by venous channels was noted. Vessels connecting the mucoperiosteum with the cranial sinuses and their tributaries may become the site of thrombophlebitis. This is the usual manner in which infection spreads from the frontal sinus to the dura. Extension of infection from the sphenoid and ethmoid sinuses by this means is rare.

5. Congenital bony dehiscences may permit direct spread of infection.

6. Spread through a persistent craniopharyngeal pouch occurred in one case.

CASES STUDIED.

During the past two years five patients have been observed at the Mayo Clinic who developed acute purulent meningitis following intranasal operation for removal of nasal polyps. Ethmoidectomy was performed in each case. All patients recovered.

No information is available to demonstrate the mode of infection of the intracranial space. One fact stands out in all

cases: headache, the sentinel sign of impending intracranial complication, was noted within 36 hours after the operation, and in four of the five cases the diagnosis of meningitis was definitely established within this period. The rapidity with which intracranial infection developed, in our opinion, indicates that infection reached the intracranial space by either *1.* trauma to the roof of the ethmoid or the cribriform plate, or *2.* extension along a preformed pathway, the perforating veins communicating with meningeal veins, the perivascular lymph channels, the olfactory nerves or the perineural lymph spaces. It does not seem reasonable that meningitis could develop as the result of osteomyelitis and subdural abscess in so short a time. The absence of signs of thrombosis of major intracranial venous sinuses indicates that extension did not occur by way of veins communicating with these sinuses.

The surgical technique employed in operations in these cases did not differ from that used in other similar cases in which intracranial complications did not develop. Presenting polyps were removed with the cold snare. The ethmoid cells were broken down with curettes and tissues removed with alligator biting forceps, care being taken not to apply force in a direction toward the roof of the ethmoid cavity. Evidence of chronic sinal infection was present in these cases. Infection is the rule when polyps are of long standing and its presence does not indicate that risk of operation in these cases was unusual. A pack of petrolatum gauze was placed in the nasal chamber in two of the five cases to maintain the position of radium applicators. The radium was removed after three hours, the packs after 24 hours. We have no adequate explanation for the occurrence of intracranial infection in this group of patients.

REPORT OF CASES.

Case 1 (see Fig. 1): A white man, aged 72 years, was examined Dec. 12, 1946. He had undergone nasal polypectomy in 1925 and 1938. In 1931, submucous resection of the nasal septum and a Denker operation on the right antrum had been performed.

On examination at the clinic, large polyps arising from the region of the middle meatus were present in each nasal chamber. No pus was seen in

the nose. Roentgen examination of the paranasal sinuses revealed thickened membrane and polypoid hyperplasia.

On Dec. 24, 1946, with the patient under local anesthesia obtained by use of cocaine and procaine hydrochloride, numerous large polyps were removed and the ethmoid cells were exenterated. Radium was applied to the ethmoid area on each side.

On Dec. 25, 1946, the patient complained of generalized headache and malaise. Examination revealed slight rigidity of the neck. The temperature rose rapidly to 105° F. Spinal puncture was done and cloudy fluid was withdrawn. The spinal fluid contained 4,100 polymorphonuclear leuco-

MENINGITIS FOLLOWING NASAL POLYPECTOMY

Days	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Temperature		Admission	Operation													
Penicillin		120	300	360	360	360	360	360	360	360	360	360	360	360	360	
Sulfa, total		4.0	12.0	6.0	3.0	9.0	9.0	6.0	6.0	6.0	6.0	6.0	6.0	6.0	6.0	
Sulfa level				17.4	20.3	9.3		16.5			17.4		13.1		14.6	
Penicillin intrathecal				20		20		20								
Whole blood									500							

Fig. 1. Temperature chart, Case 1.

cytes per cubic millimeter. Culture revealed Diplococcus pneumoniae, group D.

Treatment consisted of administration of sodium sulfadiazine and sodium sulfamerazine by the intravenous route. These drugs were given by the oral route when the patient was able to cooperate. The concentration of sulfonamide was 17.4 mg. per 100 cc. of blood on Dec. 26, 1946, and was maintained at more than 15 mg. per 100 cc. until Jan. 11, 1947. Following this, the dosage was reduced and use of the drug was discontinued on Jan. 15, 1947.

The sodium salt of penicillin was administered in doses of 30,000 Oxford units every three hours by the intramuscular route. In addition, the patient was given 20,000 Oxford units of penicillin by intrathecal injection on Dec. 26, 27 and 28, 1946.

On this regimen the temperature subsided gradually and reached the normal level Jan. 5, 1947.

On Jan. 7, 1947, symptoms of acute infection of the abdomen developed. Laparatomy was performed and an acute gangrenous gallbladder with stones was removed. Recovery ensued and the patient was dismissed Feb. 4, 1947.

Case 2 (see Fig. 2): A white man, aged 45 years, was admitted Feb. 4, 1947. He complained of nasal obstruction which had been present all his life. History did not reveal symptoms of allergy.

Examination revealed polyps in each middle meatus. Polyps blocked the choana on the left side. Thick pus was present in each nasal chamber. Roentgen examination revealed thickened membrane in all the sinuses.

Feb. 10, 1947, with the patient under local anesthesia, bilateral intranasal operation was performed. Polyps were removed from the middle meatus on each side. The anterior ethmoid cells were exenterated. The intercellular septa were very thin. The cells contained inflamed mucosa and pus. Nasoantral windows were made in each inferior meatus. The nose was not packed.

On Feb. 11, 1947, at noon, the patient noted headache and had a chill with sharp rise of temperature to 104° F. He vomited and had an involuntary bowel movement in bed. At 6 p. m., slight nuchal rigidity was noted. On Feb. 12, the typical picture of meningitis was present. Spinal puncture was done and cloudy fluid was withdrawn. Laboratory examination of the spinal fluid revealed 5,700 cells per cubic millimeter. Diplococcus pneumoniae, group D, was isolated by culture.

Treatment for meningitis was started on Feb. 11, 1947. Penicillin was administered by the intramuscular route in doses of 20,000 Oxford units every three hours. This dose was increased on Feb. 13, 1947, to 50,000

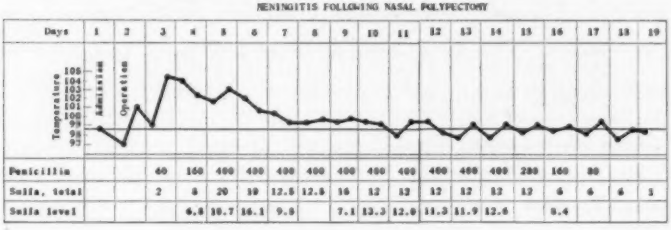

Fig. 2. Temperature chart, Case 2.

units every three hours. This treatment was discontinued on Feb. 25, 1947.

Sulfadiazine was administered orally in doses of 1 gm. every four hours on Feb. 11, 1947; this dose was increased to 2 gm. every four hours on Feb. 12 and its use was continued to Feb. 24, when it was reduced to 1 gm. every four hours. This dosage was supplemented by the daily administration of 10 to 12.5 gm. of sodium sulfadiazine by the intravenous route from Feb. 13 to 16. Despite these large doses, the concentration of sulfadiazine in the blood exceeded 11 mg. per 100 cc. on only one of these days, Feb. 14, when it was 16.1 mg. Therapy was discontinued Feb. 27, 1947.

The temperature subsided by lysis and reached an approximately normal level on Feb. 16, 1947.

A complication which caused considerable concern was the development of marked rigidity of the abdomen 24 hours after the onset of symptoms of meningitis. In view of history which was suggestive of peptic ulcer, perforation of peptic ulcer was suspected. Because of the patient's poor condition a nasal tube was inserted into the duodenum, and continuous suction was applied. The patient at no time suffered from pain in the abdomen, or exhibited tenderness consistent with perforated peptic ulcer. Rigidity of the abdominal muscles subsided after three days.

A second complication which explains the difficulty of maintaining the desired concentration of sulfonamide in the blood was the development of symptoms suggesting diabetes insipidus. The specific gravity of the urine before operation was 1.027, and the day following operation was 1.020. After the onset of meningitis the specific gravity of the urine gradually decreased and reached the low level of 1.001. The patient exhibited marked thirst and drank large quantities of water. After he was allowed out of bed he filled his own pitcher so that fluid charting was inaccurate. This condition improved after recovery.

The patient was dismissed from the hospital on Feb. 27, and from the clinic on March 6, 1947. He returned Dec. 1, 1948. Mucopus was present in the nose. No polyps were noted. Specific gravity of the urine was 1.020.

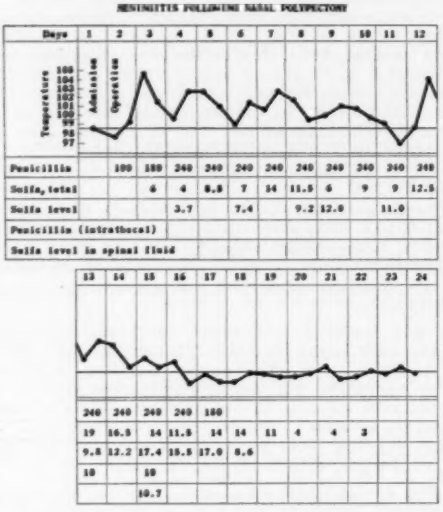

Fig. 3 . Temperature chart, Case 3.

Case 3 (see Fig. 3): A white man, aged 48 years, registered July 18, 1947. He complained of nasal obstruction and sneezing when in contact with farm dusts. In January, 1947, he caught cold and had suffered from asthma ever since. A diagnosis of asthmatic bronchitis was made. Nasal examination revealed bilateral polyps in the middle and superior meatuses. Pus was present in the choanae on repeated observations. Lavage of the antra was done with clear return. Roentgen examination showed thickened membrane in all the paranasal sinuses. Skin sensitization tests showed slight reactions to a few inhalant allergens.

On July 24, 1947, with the patient under local anesthesia, an intranasal operation was performed. Frank pus was found in both sphenoid sinuses and in the right maxillary sinus. The walls of the anterior and posterior

ethmoid cells were extremely thin and decalcified. The ethmoids were exenterated. Radium was applied to the sphenoethmoid recess on each side and maintained in position for three hours by a pack of petrolatum gauze. The pack was removed 24 hours later.

On July 25, 1947, the patient complained of severe generalized headache and the temperature rose abruptly to 104.6° F. He had no chills and no vomiting. Slight rigidity of the neck was noted. On July 26, signs of meningitis were well developed. Spinal fluid withdrawn contained 6,378 polymorphonuclear cells per cubic millimeter. Diplococcus pneumoniae was isolated by culture.

Treatment consisted of sulfadiazine administered by mouth. The initial dose was 4 gm.; the second dose, four hours later, was 2 gm., and subsequently doses of 1 gm. were given each four hours. After 24 hours the concentration of sulfadiazine was only 3.7 mg. per 100 cc. Dosage was increased to 1.5 gm. every four hours, which gave concentrations in the blood of 7.4 mg. per 100 cc. The patient did not tolerate higher doses of sulfonamide by mouth. Oral medication was supplemented by intravenous injection of 5 to 10 gm. of sulfadiazine daily, beginning Aug. 3, 1947, with resulting concentrations in the blood of between 12 and 17 mg. Penicillin was administered by intramuscular injection of 20,000 units every three hours, beginning at the time of surgery. The dose of this drug was increased to 30,000 units every three hours at the onset of signs of meningitis. Spinal fluid was removed on July 26 and 31 and on Aug. 3, 4, 6 and 12, 1947.

The patient made slow but steady improvement from the onset of his symptoms until Aug. 3, 1947, when headache, vomiting and nuchal rigidity recurred. At this time sodium sulfadiazine was given by intravenous injection. Improvement again followed, the temperature reaching normal Aug. 12, 1947. He was dismissed from the hospital Aug. 15 and from the clinic Aug. 19, 1947.

The patient was observed in October, 1947. Pus was present in the upper part of the right nasal chamber. This diminished after local treatment. No polyps were noted. The patient had had no severe attack of asthma since his operation. He was again observed Sept. 1, 1948; at this time he reported that he was having mild symptoms of asthma almost daily, but no severe attacks. A small polyp was present in the left nasofrontal duct and pus was observed around the polyp. The polyp receded after removal of the pus with a suction cannula.

Case 4 (see Fig. 4): A white man, aged 41 years, was examined Jan. 5, 1948. His presenting complaint was "gas on the stomach."

Examination of the nose was done because he complained of drainage of pus from the nose. Rhinoscopy revealed atrophy of the nasal mucosa. Multiple polyps which were bathed in pus were seen to arise from each middle meatus. Roentgenograms showed thickened membrane and possibly polyps in each ethmoid. Lavage of the antra returned mucopurulent material from the right, but the return from the left was clear.

On Jan. 23, 1948, an intranasal operation was done with the patient under local anesthesia. The anterior ethmoid cells were exenterated. The ethmoid cells contained hyperplastic mucosa and pus. The nasoantral wall was removed in each inferior meatus. Polyps were present in the left antrum.

On Jan. 25, 1948, the patient complained of severe bifrontal headache, which he stated had begun the evening before. Signs suggesting meningitis were noted. Cloudy spinal fluid was withdrawn. The cell count was

inaccurate, due to clumping of the leucocytes. Smear and culture did not reveal micro-organisms.

On the evening of Jan. 25, 1948, a rise in temperature was noted, flexion of the neck was slightly limited, and Kernig's sign was slightly positive on the right side. On Jan. 26, clinical signs of meningitis were definite, although the temperature was lower and the patient felt much better.

Sulfonamide therapy consisted of sulfamerazine given orally. Four gm.

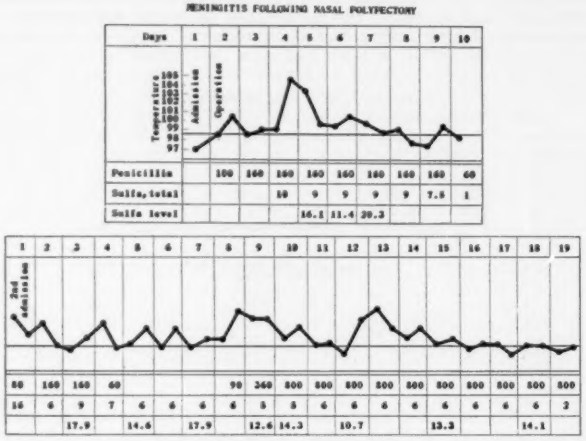

Fig. 4. Temperature chart, Case 4.

were given at the first dose, followed by four doses of 2 gm. each and continued with 1.5 gm. each four hours thereafter. This dosage resulted in a concentration of 16.1 mg. of sulfonamide per 100 cc. of blood at the end of 24 hours, and the concentration was maintained between 14.6 and 20.3 mg. Penicillin was administered concurrently.

This patient responded rapidly to treatment. The temperature dropped to 100° F. one day after the initial rise and became normal Jan. 29. The patient was dismissed from the hospital Jan. 31, 1948. He was instructed to take 1 gm. of sulfamerazine four times daily.

The headache recurred the evening of Feb. 1, 1948, and the following morning projectile vomiting occurred. He was readmitted to the hospital. The temperature was 101° F. Neurologic examination and examination of the ocular fundi gave negative results. During the following eight days several slight rises of temperature were noted. The patient complained of moderate headache. Examinations repeatedly gave negative results. Brain abscess or localized meningitis was suspected. The patient remained in the hospital until Feb. 21, 1948. At this time his temperature had remained normal for seven days. Observation was continued until Feb. 28, 1948. At the time of dismissal he had had no headache or other complaints for two weeks.

Treatment following readmission to the hospital consisted of intramuscular administration of 20,000 units of penicillin every three hours from Feb. 2 to 5, 1948, when the dose of penicillin was increased to 100,000 units every three hours. Sulfamerazine by mouth, and sodium sulfadiazine by intravenous injection were given in amounts adequate to maintain concentrations of more than 12 mg. of the drug per 100 cc. of blood. Following his dismissal from the hospital, he was given 600,000 units of procaine penicillin intramuscularly daily until his dismissal from the clinic.

Case 5 (see Fig. 5): A white man, aged 61 years, was first examined at the clinic on June 2, 1948. He complained of nasal obstruction of 15 years' duration. Nasal polyps had been removed in 1943, and again in

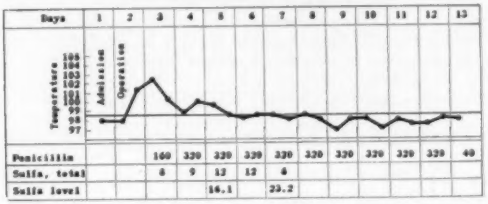

Fig. 5. Temperature chart, Case 5.

1947. He had suffered from headache in both the supraorbital and occipital regions. The occipital headache was relieved by massaging the muscles of the neck. He sneezed a few times each day.

Examination revealed multiple polyps in each side of the nose. A large polyp protruded from the choana on the right side. The sinuses could not be entered by probing. Roentgenograms revealed thickened membrane and secretion in all sinuses of the anterior group. Those of the posterior group were clear. General physical examination gave negative results. Systolic blood pressure was 120 mm. of mercury; diastolic pressure was 85 mm.

An intranasal operation was done June 5, 1948. Local anesthesia was used. Multiple polyps were removed from the right ethmoid area by snare and biting forceps. The walls of the ethmoid cells were thickened and the cells were filled with polyps. The ethmoid was exenterated. A nasoantral window was made and a huge polyp was removed from the right antrum. The mucosa forming the ostium of the sphenoid sinus showed polypoid degeneration. The ostium was enlarged. The mucosa of the sphenoid was normal. On the left side polyps were smaller. Findings in the ethmoid capsule were similar to those on the right. Polyps were removed and the ethmoid was exenterated. The left sphenoid sinus was large and presented through a dehiscence in the root of the middle turbinate. At this point the patient went into a state of collapse and the operation was discontinued. No packs were used.

The episode at the end of the operation consisted of paralysis of the right side of the body and aphasia for speech. The patient was observed closely. Motion of the extremities returned one hour and speech returned

two hours after the onset of the complication. This episode was interpreted as being due to vascular spasm.

The morning following operation the patient complained of generalized headache. Speech was clear and there was no paralysis. He complained of pain in the muscles of the neck. Two hours later, he became stuporous and the temperature rose to 102.8° F. Moderate rigidity of the muscles of the neck was present. Spinal puncture was done. The spinal fluid was turbid and contained leucocytes too numerous for accurate count. Culture revealed nonhemolytic streptococci.

Penicillin in doses of 40,000 units was administered every three hours by intramuscular injection. At the onset of symptoms of meningitis, 5 gm. of sodium sulfadiazine were administered intravenously. Following this initial dose, the patient was given 6 gm. of sulfadiazine, 3 gm. of sulfathiazole and 3 gm. of sulfamerazine daily. Sulfonamide medication was discontinued after four days because of crystalluria. Concentrations of sulfonamide in the blood were 16.1 and 23.2 mg. per 100 cc.

The patient made excellent progress. His temperature became normal on the third postoperative day and remained normal. Headache and numbness of the right leg persisted for three days. He was dismissed from the hospital June 16, 1948, 10 days after the onset of meningitis. He was dismissed from observation June 21, 1948.

TREATMENT.

Study of this small group of cases gives support to the basic rules for chemotherapy of acute infection. These rules, stated briefly, are treat early, treat adequately and treat long enough. In all cases administration of a drug of the sulfonamide series was instituted immediately after the onset of intracranial complication was recognized. Variations in the initial dosage of the drug and in the time required to achieve adequate concentration of the drug in blood are reflected graphically in the response of the patient to treatment. In two cases in which treatment was reduced too early, meningeal symptoms recurred. The dosage of sulfonamide should be controlled by determinations of the concentration of the drug in the blood. In Case 2, because of the tremendous fluid exchange, concentrations of sulfonamide remained low despite seemingly adequate doses of the drug. In Case 5, the same dose resulted in high concentrations of the drug in the blood.

Penicillin is of limited value in prevention or treatment of postoperative meningitis of nasal origin. Three of the five patients in this series received penicillin from the time of operation. Penicillin does not penetrate nerve tissue or the

meninges; however, penicillin is of value in reducing the continued spread of infection from the primary focus to the meninges and for this reason should be administered to these patients.

COMPLICATIONS.

Symptoms of acute abdominal disease developed during the course of the illness in two cases. It is fortunate that in the case in which laparotomy was necessary, the abdominal infection developed after recovery from the meningitis. Certainly risk of performing a major surgical procedure on a patient suffering from severe meningitis would be high.

Rigidity of the abdominal wall may be due to meningism. The occurrence of this symptom in a patient severely ill and semistuporous creates a diagnostic problem of the first magnitude. The decision in favor of conservative management in Case 2 was a fortunate one.

Development of symptoms suggesting diabetes insipidus in Case 2 which were transient in duration indicates pituitary dysfunction resulting from inflammation. The influence of these symptoms on treatment of the infection has been noted. It is probable that strict limitation of intake of fluid would have been valuable in this case.

COMMENT.

From the study of these five cases certain points should be emphasized in summary:

1. Meningitis is a definite risk in operations on the ethmoid for removal of nasal polyps.

2. Penicillin administered postoperatively does not prevent meningitis.

3. The sulfonamides are the most valuable drug for treatment of meningitis.

4. Successful results require early, adequate and prolonged sulfonamide therapy.

5. Dosage of sulfonamides should be controlled by determination of the concentration of the drug in the blood.

6. Complications of meningitis may involve systems outside the central nervous system.

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USE OF THE ALNICO MAGNET IN BRONCHOSCOPY AND ESOPHAGOSCOPY.

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Denver, Colo.

A recent addition to the bronchoscopist's armamentarium in the form of the alnico magnet has proven of great value for the removal of certain metallic foreign bodies from the bronchi, esophagus and stomach. Lately, I have had gratifying results with this magnet in cases which previously had presented formidable foreign body problems. The facts of its adaptability, simplicity and safety may advantageously be reemphasized.

Silber, Kaplan and Epstein[1] reported its employment in 1943 for the removal of a padlock from a child's stomach. Equen[2] soon thereafter elaborated on its usefulness and described the specifications of the magnet he devised for use in the esophagus and stomach and of a smaller one for the tracheobronchial tree (see Fig. 1). The magnet for the esophagus and stomach is cylindrical, 5 cm. in length by 0.5 cm. in diameter, and is attached to a Levine tube, size 12F, perforated near its attachment to the metal to allow inflation of the stomach with air. A stylet in the Levine tube increases its rigidity.

The magnet for the trachea and bronchi is smaller, 4 cm. in length by 0.3 cm. in diameter, and is attached to an ureteral catheter.

Alnico[3] is an alloy of aluminum, cobalt, copper and iron. It is a permanent type of magnet, requires no electrical current to energize it and is said to be capable of lifting objects 60 times its own weight.

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Although the endoscopic extraction of most foreign bodies that have been aspirated or swallowed has been satisfactorily standardized, the peroral removal of objects such as open safety pins from the esophagus and stomach,[4] and straight pins from the smaller bronchi situated close to the diaphragm, is difficult and frequently fraught with danger. Where available, the use of the biplane fluoroscope has been of great

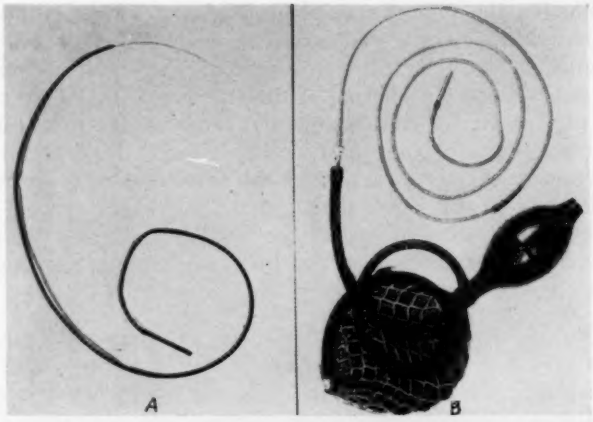

Fig. 1. Alnico magnets designed by Equen for extraction of magnetizable foreign bodies. At (A) is shown the magnet for use in the bronchi. At (B) is the magnet for use in the esophagus and stomach with attachments for insufflating air into stomach.

assistance. Unfortunately, even in some of our larger communities the biplane fluoroscope is not yet to be had. Without it, the extraction of a straight pin from a bronchus too small to permit its visualization with a bronchoscope is uncertain if not impossible.[5] It is just in this type of difficult foreign body problem that the alnico magnet has been found to have great usefulness, as subsequent case reports will illustrate.

Obviously, the object to be extracted with the magnet must be magnetizable metal and it is well to attempt to obtain a duplicate of the intruder to test for this quality. Many of the straight and safety pins now in use are made of iron. Bobby

pins, which are now so frequently swallowed, may be very dramatically extracted from the stomach with the alnico magnet. Peculiarly, a bobby pin seems to have great difficulty in leaving the stomach, and where no progress has been apparent for several days it would seem best to "fish it out" with the magnet and thus spare the patient and parents needless anxiety.

Where an open safety pin, point up, is found lodged in the esouhagus, the magnet may be inserted as a bougie and, under ordinary fluoroscopic guidance, the pin, in contact with the magnet, is directed into the stomach. By manipulations of the magnet and insufflation of the stomach with air, to allow the pin freedom of motion, the intruder may readily be flipped over so that its point now trails. It may then be drawn upwards past the cardia and up the esophagus without undue resistance. If it becomes detached at the cricopharyngeal constriction at the upper end of the esophagus, as it sometimes may, the pin can then simply be extracted by inserting an esophagoscope and grasping it with forceps.

The question may arise as to the risk involved in the maneuvers necessary to effect a change in the presentation of the open safety pin in the stomach. The answer is that it is practically *nil*, as the magnet's pull against the point of the pin would appear to be insufficient to produce a perforating or even traumatizing injury to the gastric mucosa. Experimentally flipping the open safety pin with the magnet in the palm of one's own hand will demonstrate that the magnet will become detached from the object before its point will cause pain or injury to the skin.

When confronted with the problem of extracting a straight pin from a peripheral bronchus, fluoroscopic guidance should also preferably be employed, as this will assist in directing the magnet into the proper branch bronchus. Searching and grasping for the pin with forceps should not be the primary procedure if the use of the magnet is contemplated. The magnet should be tried first, especially if the pin is point up, as with its use the point is not so apt to be caught in the bron-

chial wall. This often happens when the pin is grasped with forceps. Application of the magnet at this juncture may not succeed in removing the intruder.

CASE REPORTS.

Case 1: J. G., age six years, was seen at St. Joseph's Hospital on July 19, 1946, at the request of Dr. J. F. Harrington, because of a straight pin that had become lodged in the child's hypopharynx (see Fig. 2). Esopha-

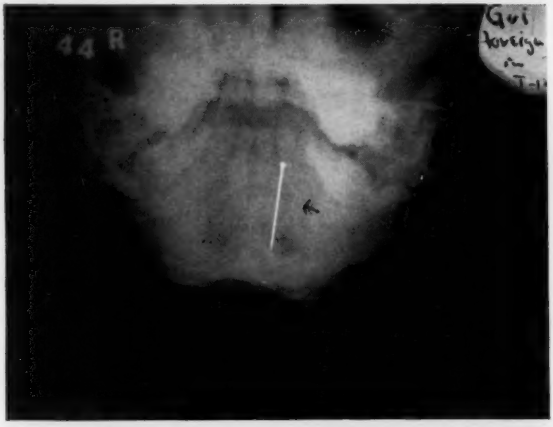

Fig. 2. Straight pin in hypopharynx of a six-year-old child (Case 1).

goscopy was prepared for a few hours later. An X-ray taken just prior to surgery showed the pin at about the previously found level. The patient was anesthetized with ether and the pin searched for, but it could not be located in the pharynx or esophagus. An X-ray of the chest and abdomen was then made with a portable unit, and the pin was found to have lodged in the lower portion of the right lung field, not far above the diaphragm (see Fig. 3). A bronchoscope was inserted, but the pin could not be visualized. The alnico magnet was then introduced through the bronchoscope and after directing it into a posterior branch bronchus of the lower lobe of the right lung, the magnet was removed and the pin was found dangling to its distal end.

Case 2: S. D., age 10 months, was seen at Children's Hospital in consultation with Dr. Rex Murphy, on Sept. 18, 1946, on account of a straight pin that had lodged in the lower lobe of the right lung. He prepared for bronchoscopy under makeshift biplane fluoroscopic guidance (using a portable fluoroscope in addition to the fixed one). He visualized the pin, point up, in a lower lobe branch and grasped it with side-curved forceps. On attempting to extract it, the point got caught in the mucosa of the

bronchial wall. I then inserted the alnico magnet but the pin could not be dislodged, apparently owing to the fact that its point was caught. After further manipulation with forceps and with the aid of the fluoroscopes, Dr. Murphy shortly thereafter was able to extract the pin.

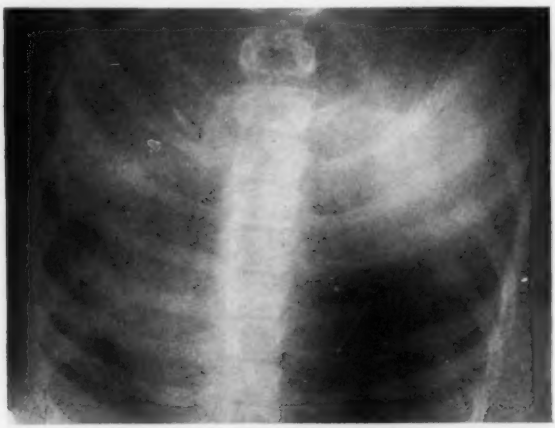

Fig. 3. Straight pin shown in Fig. 2 after lodging at base of right lung (Case 1).

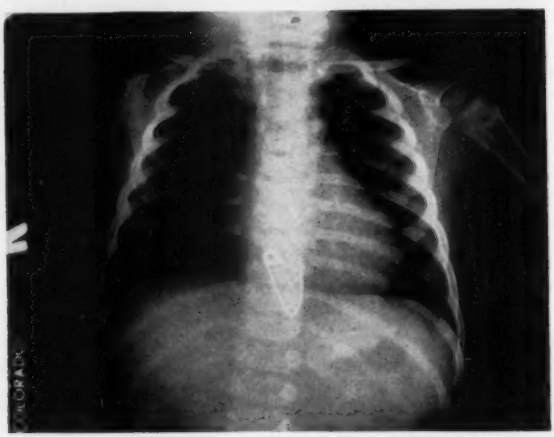

Fig. 4. Open safety pin, point up, lodged at the lower end of the esophagus in a 10-month-old child (Case 3).

Comment: Experience with this case would indicate that application of the magnet for extracting a straight pin from the lung should be the primary procedure, rather than attempting its use following manipulation with forceps, as prior to grasping it the point is less apt to be embedded in the bronchial mucosa.

Case 3: A. B., age 10 months, swallowed an open safety pin on Sept. 28, 1946. The pin with its point up was found by X-ray to have lodged near the cardiac end of the esophagus (see Fig. 4). A member of the bronchoscopic staff of the Colorado General Hospital was unsuccessful in his efforts to remove the safety pin. An X-ray taken the following day showed the pin at approximately the same level. Preparation was made

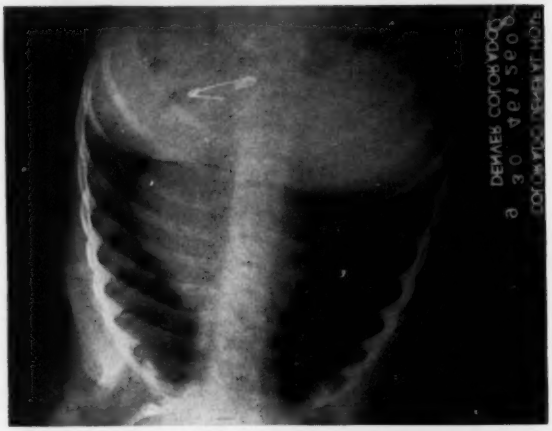

Fig. 5. Open safety pin in the stomach of a child 10 months old after spontaneously leaving its site of lodgment in the lower end of the esophagus (see Fig. 4). The pin was extracted from the stomach by use of the alnico magnet (Case 3).

to use the alnico magnet the following morning; however, the radiogram taken just prior to surgery revealed that the pin had descended into the stomach (see Fig. 5). It was felt that the possibility of the pin passing spontaneously was quite good and the contemplated procedure with the magnet was deferred on that account. Surgical consultation obtained by the pediatric staff was reported to be in favor of gastrostomy. It was my feeling that the magnet should be tried first and the following day this was done. Under ether anesthesia the magnet was introduced into the stomach. The stomach was then inflated with air and the magnet and child were so manipulated that the spring end of the pin pointed upwards.

With the point thus trailing, the pin was pulled up past the cardia, then up the esophagus as far as the cricopharyngeal constriction, where it became detached from the magnet. An esophagoscope was then inserted and the pin extracted by means of a forceps.

Case 4: P. W., age nine years, was referred by Dr. Max Ginsburg on

account of a bobby pin lodged in her stomach for four days. On Sept. 23, 1947, the patient was removed to Children's Hospital and the alnico magnet introduced into her stomach with fluoroscopic guidance. The pin easily attached itself to the magnet and was removed.

Case 5: J. S., age three years, was referred by Dr. Emanuel Friedman for the magnetic removal of a bobby pin which had been lodged in the child's stomach for nine days. Repeated X-rays had shown that the pin had not succeeded in passing the pylorus (see Fig. 6). She was removed

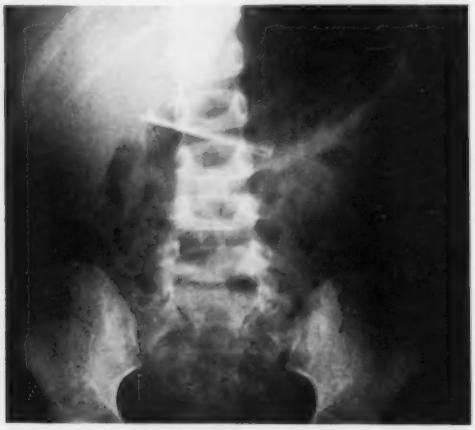

Fig. 6. Bobby pin in the stomach of a three-year-old child, nine days after it had been swallowed. The patient was given a Seidlitz powder in order to distend the stomach with gas, as it could not otherwise be definitely determined that the bobby pin was actually in the stomach and not in a coil of intestine. Note that the pronged end of the pin is caught in the pyloric ring (Case 5).

to Children's Hospital on Dec. 6, 1947; the alnico magnet was passed into her stomach under fluoroscopic guidance and the bobby pin promptly extracted.

CONCLUSIONS.

1. The alnico magnet is a useful addition to the endoscopist's armamentarium as an aid in the removal of magnetizable foreign bodies.

2. It is a simple, safe instrument and its use is relatively free from danger.

3. The alnico magnet may render great assistance in the

extraction of open safety pins from the esophagus and stomach, and straight pins from the periphery of the lung.

4. Fluoroscopic aid plays an essential part in the manipulation of the alnico magnet.

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227 Sixteenth Street.

INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY, LONDON, 1949.

The British Association of Otolaryngologists is organizing the Fourth International Congress of Otolaryngology, to be held in London from July 17 to July 23, 1949. There will be further meetings, for those who wish to go, at Oxford, Cambridge and Edinburgh on July 25 and 26. It is hoped that a full academic program will be arranged, and also various social functions.

The secretaries of the National Otolaryngological Societies have been circularized and asked to send a list of their members for individual notification. Should any association not receive this letter, they should communicate with the General Secretary, F. C. W. Capps, F.R.C.S., 45, Lincoln's Inn Fields, London, W.C. 2.

BIFID EPIGLOTTIS. REPORT OF A CASE.*

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Malformations of the larynx are very rare and among these the more frequent ones are those of the epiglottis (Beck and Schneider[1]).

Apart from the anomalies in shape, such as the exaggerated infantile type and the broad based flaccid epiglottis, there are only two other malformations reported: the bifid epiglottis and absence of the epiglottis.

In the case of bifid epiglottis the organ is split in midline to a lesser or greater degree, from a mere indentation of the free border to that of a complete split extending down to the petiolus. In some of these cases the splitting of the cartilage would be more extensive on anatomical examination than it would appear on clinical examination. Bifid epiglottis is of frequent occurrence only if one includes small indentations of its free border (Weingaertner[2]).

To the 16 cases found in the literature (Manifold,[3] Mackenzie,[4] Schreiber,[5] Calmann,[6] Refslund,[7] Henke,[8] Weingaertner,[2] Culp,[9] Jackson[10]), the report of an additional case seems to be of interest.

CASE REPORT.

On Feb. 5, 1947, K. P., a baby girl, was admitted to the Babies' Hospital at the age of six weeks, with the complaint of persisting respiratory stridor and dyspnea.

Family History: Mother, 25, and father, 28, are both in excellent health, neither one presents any malformation, nor is there any malformation known to have existed in either family for three generations. Examina-

*From the Department of Otolaryngology of the Columbia University College of Physicians and Surgeons and the Presbyterian Hospital. The author was assisted by a grant from the Hayden-Coakley Fund.

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tion of the larynx of both the father and the mother proved to be essentially normal.

Physical Examination on Admission: Fairly well developed and nourished white, one-month premature female baby with slight inspiratory stridor and retraction of the suprasternal spaces. The baby has a good cry and is alert.

The general physical examination is negative except for an extra digit on each hand (ulnar) and foot.

Laboratory findings, including X-rays of the esophagus, were all essentially normal.

On Feb. 11, 1947, because of continued respiratory difficulty, a tracheotomy was performed under local anesthesia. Except for a slight rise in temperature which lasted two days, the postoperative course was uneventful. Shortly afterwards a direct laryngoscopy showed that the epiglottis

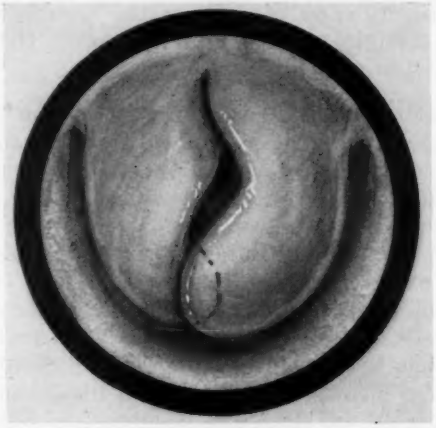

Fig. 1. Drawing of the epiglottis as seen through direct laryngoscopy. The laryngeal chink and most of the aryepiglottic folds are masked by the overlying epiglottic halves. Note the left epiglottic half which lies on a deeper plane and whose tip is partly covered by the right half.

was completely split in midline, from its free edge down to its base, forming two equal parts. Both halves appeared to be very lax. The left half was seen to lie deeper than the right one, and the tip of the left half was somewhat obscured by the overriding right half (see Fig. 1). On inspiration, both halves of the epiglottis and the aryepiglottic folds were seen to be sucked into the glottic chink. The glottic chink appeared to be reduced in size, and a No. 16 bougie could not be passed. Subsequent laryngoscopies showed essentially the same picture, and little if any stiffening of the epiglottis could be noted.

It was at first thought that age might remedy this laxness and that the epiglottis might regain some of its tonicity. After observing the baby

for some nine months, it became evident that such would not be the case, and it was, therefore, decided to amputate the epiglottis.

On Dec. 16, 1947, under general ether anesthesia, the epiglottis was exposed by suspension laryngoscopy and both halves of the epiglottis were amputated near the base. Following operation, there appeared to be an increase in the amount of secretions, but no postoperative bleeding was encountered — swallowing was essentially normal. On Dec. 24, 1947, the tracheotomy was half corked, and on Jan. 10, 1948, was fully corked. On Jan. 19, 1948, the tracheotomy tube was removed and tracheotomy was allowed to close.

Laryngoscopy on Jan. 9, 1948, revealed a well healed epiglottic stump which now was not sucked in with inspiration. On Jan. 26, 1948, the baby was discharged from the hospital and has remained well ever since. The baby was last seen and laryngoscoped in May, at which time no change was noted. The tracheostomy has closed over, leaving a fairly large scar, which it is intended to remove by plastic repair at a later date.

Among the 16 cases reported in the literature, there are few with a complete splitting of the epiglottis. Only two cases are described in any detail.

Weingatertner,[2] in 1920, describes the case of an adult on whom a mirror examination revealed a split of the epiglottis extending from its free border to its inferior third. The inferior third or base of the epiglottis apparently presented a split cartilage whose halves were covered over by normal mucosa. It is interesting to note that this patient also presented an extra digit on each hand and foot. In his case report, Weingaertner states that only three other cases are to be found in literature. He mentions Henke's four cases, but somehow fails to include them, and, as for Schreiber's case which he also mentions, he omits it on the grounds that his description is not too clear, and Weingaertner refused to recognize it as a case of bifid epiglottis. On reviewing Schreiber's[5] case, however, it appears unquestionably to be one of bifid epiglottis.

Of Henke's[8] eight cases, four were admittedly marginal defects and are not counted, and of the remaining four cases, only in two was the split seen to extend down to the base.

The only case examined histologically is one reported by Culp.[9] Here the bifid epiglottis was an accidental postmortem finding in a one-and-one-half-year-old baby girl. On macroscopic examination the epiglottis was split for a distance of

4 mm. from its free border. On histological examination, however, the cartilage was found to be split for a greater distance and extended down to its base. The two halves of cartilage at the base were united by dense connective tissue. Each half presented two separate chondrification centers.

Schreiber's[5] case, contrary to Weingaertner's opinion, is thought to represent a case of bifid epiglottis.

Refslund's[7] case can hardly be called a bifid epiglottis, as apparently only the cartilage was split and not the covering mucosa.

Calmann's[6] case not only presented one midline split, but three, thus dividing the epiglottis into four parts.

Manifold and Mackenzie's original articles were not to be found in New York or the Surgeon General's Library.

The explanation of this malformation is not easy, mainly because of the fact that there is no unanimity among the authors concerning the development of the epiglottis.

The first appearance of the larynx is found in 6 mm. embryos (25 to 26 days) (Soulie and Bardier,[11] J. E. Frazer[12]). At this time the respiratory groove is transformed into the laryngotracheal duct by lateral mesenchymal ingrowths. At this stage also appears a large medial swelling on the anterior wall of the duct, the so-called furcula of His. All authors agree that the furcula of His takes part in the formation of the epiglottis through its posterior portion and in the formation of the tongue through its anterior portion. Two points are controversial, however: whether the furcula is of bilateral origin or not, and whether the furcula is the sole anlage of the epiglottis.

According to Frazer,[12] whose work is about the most recent on the subject, the epiglottis is derived solely from the furcula, which is, from the very beginning, of impaired and medial origin, the fourth branchial arches playing no part in the formation of the epiglottis and only participating in the formation of the aryepiglottic folds.

On the other hand, Soulie and Bardier concluded that the furcula had a bilateral origin. These authors noted in very young embryos (6 and 7 mm.) that as soon as the furcula made its appearance, this swelling always presented a small indentation of both its superior and inferior borders, thus attesting to a bilateral origin.

Roth,[13] studying older embryos, came to the conclusion that the epiglottis had both a medial and a bilateral origin, because on his embryos he found a small groove just off midline on both sides, which grooves divided the swelling into three distinct parts, one medial and two lateral. This theory would render the explanation of the case described by Calmann much easier.

Kallius,[14] noting the same findings, explained that the medial portion only contained cartilage and developed into the epiglottis, whereas the lateral portions, devoid of cartilage, gave rise to the aryepiglottic folds.

Soulie and Bardier[11] explain this medial portion as the resultant of the fusion, in midline, of the two lateral portions.

A bifid epiglottis could easily be explained by assuming a bilateral anlage of the furcula, either through absence or incomplete fusion of the lateral masses. To a certain extent these cases of bifid epiglottis could be used as an indirect proof for the assumption of a bilateral origin of the epiglottis. Additional proof can be gained by the fact that in certain animals a bifid epiglottis is known to exist, such as in the sea-lion (Negus[5]). In Echidna, the epiglottis, although not showing any visible split, contains, nevertheless, two separate halves of cartilage which are united in midline by dense connective tissue (Negus).

The final answer to this question will have to be found in further studies on the subject of the development of the epiglottis.

SUMMARY.

A case of bifid epiglottis is reported, in which both halves

were very flaccid and caused laryngeal obstruction which necessitated a tracheotomy, and later on, amputation of the epiglottis. Sixteen cases are mentioned in the literature. Eleven of these cases are not considered to represent true bifid epiglottis.

The embryology of the epiglottis is discussed in an attempt to explain this malformation and the assumption of a bilateral eral origin of the epiglottis with absence or incomplete fusion between the two parts most easily explain the bifid epiglottis.

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FENESTRATION OF THE LABYRINTH.*

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BOOK REVIEW.

Phylogenesis of the Ear. By Louis Guggenheim, M.D., Associate Professor of Otolaryngology ,University of Southern California; formerly Assistant Professor of Otolaryngology, Washington University School of Medicine, St. Louis; formerly Research Associate, University of California, Los Angeles; Fellow of the American Medical Association, American College of Surgeons, American Otological Society, American Laryngological, Rhinological and Otological Society, Inc., American Academy of Ophthalmology and Otolaryngology, etc. Two hundred seventy-one pages and Index, with 196 illustrations. Culver City, Calif.: Murray & Gee, Inc., 1948. Price $12.50.

Dr. Guggenheim's interest in the comparative aspects of the ear is to be highly commended. There are certainly few otologists who would undertake a similar task.

It is unfortunate that the book is too superficial in its scope to be of much, if any, scientific value. This is apparently not the purpose for which it is intended, because, in the Foreword, it is mentioned that its contents are primarily dedicated to the otologist. Aside from the fact that the book is pleasant reading, it lacks in detailed information and cannot be considered as a valuable reference book.

The first half of the book is devoted to the phylogenetic development of the ear in invertebrates. It is apparent that standard elementary textbooks of zoology and comparative biology have furnished the chief sources of information for these chapters. Whole phyla are disposed of in one or two paragraphs, although further detail must exist in the literature. On page 51 there are three phyla discussed.

There should have been some attempt made to consider the extensive literature which exists on the balancing mechanisms in lower forms. He attributes the efficiency of the balancing mechanism in protozoa to protoplasmic density, which is certainly not the case.

A considerable amount of the 277 pages is given up to illustrations. Many of these are good, but there are also poor ones. The photomicrographs in several instances are indistinct The descriptions accompanying the illustrations are sometimes incomplete. For example, in showing a rat embryo section it is labeled "comparable to a human embryo of two and a half months." Although the text mentions the age of the rat embryo, it is not included with the illustration.

The large type and number of illustrations make the text extremely easy to read. W. P. C.

MISSISSIPPI VALLEY MEDICAL SOCIETY
1949 ESSAY CONTEST.

The Ninth Annual Essay Contest of the Mississippi Valley Medical Society will be held in 1949. The Society will offer a cash prize of $100.00, a gold medal, and a certificate of award for the best unpublished essay on any subject of general medical interest (including medical economics and education) and practical value to the general practitioner of medicine. Certificates of merit may also be granted to the physicians whose essays are rated second and third best. Contestants must be members of the American Medical Association who are residents and citizens of the United States. The winner will be invited to present his contribution before the Fourteenth Annual Meeting of the Mississippi Valley Medical Society to be held in St. Louis, Mo., Sept. 28, 29, 30, 1949, the Society reserving the exclusive right to first publish the essay in its official publication — the *Mississippi Valley Medical Journal* (incorporating the *Radiologic Review*). All contributions shall be typewritten in English in manuscript form, submitted in five copies, not to exceed 5,000 words, and must be received not later than May 1, 1949. The winning essays in the 1948 contest appear in the January, 1949, issue of the *Mississippi Valley Medical Journal* (Quincy, Ill.).

Further details may be secured from Dr. Harold Swanberg, Secretary, Mississippi Valley Medical Society, 209-224 W. U. U. Building, Quincy, Ill.

THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

The annual meetings of the Triological Society and the Broncho-Esophagological Association will be held concurrently at the Drake in Chicago on April 18, 19 and 20, 1949. A joint meeting of these two societies will take place Tuesday morning, April 19. On the other days the Triological Society will meet in the mornings and the Bronchoscopic Society in the afternoons.

We also call your attention to these dates and places:

American Board of Otolaryngology—May 11-14, 1949, New York City.

American Laryngological Association — May 16-17, 1949, New York City.

American Otological Society—May 18-19, 1949, New York City.

In order to facilitate the coordination of national meetings, the Bronchoscopic, the Otological, the Laryngological and the Triological Societies are considering a five-year plan of dates and places. By adoption of such a scheme we hope to reduce the traveling mileage and the amount of time away from our offices. For additional information, write to Dr. C. Stewart Nash, Secretary, 708 Medical Arts Building, Rochester 7, N. Y.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

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Acousticon Model A-100.
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Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac; Beltone Harmony Mono-Pac.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.
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Gem Hearing Aid Model V-35.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Type K; Maico Atomeer.
Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

1947—Mears Aurophone Model 98; Mears Aurophone Model 200.
Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver).
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Otarion Model A-1; Otarion Model A-3; Otarion Models A-4 J and S; Otarion Model E-1; Otarion Model E-1S; Otarion Model E-2.

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Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

Solopak Hearing Aids.

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Sonotone Audicles No. 530, No. 531 and No. 533; Sonotone Model 600; Sonotone Model 700; Sonotone Model 900.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.
Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Telex Model 22; Telex Model 97; Telex Model 612; Telex Model 900; Telex Model 1020; Telex Model 1550.
Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonemaster Model Royal.
Manufacturer: Tonemasters, Inc., 1627 Pacific Ave., Dallas 1, Tex.

Trimm Vacuum Tube No. 300.
Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A."
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vactuphone Model 3.
Manufacturer: Allen-Howe Electronics Corp., Salem, Mass.

Western Electric Orthotronic Model; Western Electric Model 63; Western Electric Model 64; Western Electric Models 65 and 66.
Manufacturer: Western Electric Co., Inc., 120 Broadway, New York 5, N. Y.

Zenith Radionic Model A-2-A; Zenith Radionic Model A-3-A; Zenith Radionic Model B-3-A; Zenith Model 75.
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All of the accepted hearing devices employ vacuum tubes.

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